

**ANGIOGRAPHIC SEVERITY OF CAD IN PATIENTS WITH
ACUTE CORONARY SYNDROME IN CORRELATION TO
THEIR GLYCEMIC STATUS**

**DISSERTATION SUBMITTED TO
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CERTIFICATE

This is to certify that this dissertation titled “**Angiographic severity of CAD in patients with Acute coronary syndrome in correlation to their glycemc status**” is a bonafide work done by **Dr.M.GIRISH DEEPAK**, done under my direct guidance and supervision in the department of cardiology, PSG Institute of Medical Sciences and Research, Coimbatore in partial fulfillment of the regulations of **The Tamil Nadu Dr. M.G.R Medical University** for the award of DM degree in cardiology.

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DECLARATION

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INTRODUCTION

Atherosclerotic vascular diseases, which comprises coronary heart disease and cerebro-vascular disease is a major global health burden. They constitute 21.9 per cent of total deaths globally and are projected to increase further to 26.3 per cent by 2030¹.

The prevalence of diabetes is a global health burden. The overall prevalence is expected to rise from 285 million in 2010 to 438 million by the year 2030². While diabetes poses a huge economic burden to all nations, developing countries bear the highest burden since more than 80% of cases occur in these countries.

Diabetes is considered as an independent risk factor for coronary artery disease and cardio vascular diseases. As per NCEP ATP III guidelines, diabetes is considered as a coronary artery disease equivalent³.

Diabetes affects the endothelium and the glycation products get denatured and accelerate the process of atherosclerosis. Diabetic patients when compared to non-diabetics have increased risk of developing vascular complications and have two to four fold risk of developing coronary artery disease (CAD)⁴. They are more likely to develop vascular complications, affecting all the major organs of body. Cardiovascular diseases constitute one of the major cause of mortality and morbidity in diabetics, accounting for nearly 65-75% of deaths^{5,6}.

The results of Framingham study reveals a 2 fold risk of cardiovascular death in men and 4-5 fold risk of cardiovascular death in women with diabetes. They constitute the major cause of death in adult diabetic patients. Diabetics are likely to have low HDL levels than people without DM. Low HDL levels have been strongly associated with elevated risk for CAD.

Nearly 75% to 80% of deaths in diabetic patients are due to coronary artery disease, cerebro-vascular accidents and peripheral vascular disease.

Major risk factors which contribute towards cardiovascular diseases among diabetics are hypertension, dyslipidemia, hyperglycemia and obesity. There has been a direct correlation between cardio vascular complications and chronic hyperglycemia in various interventional studies^{7,8}. The state of chronic hyperglycemia has been now measured by HbA1c, which averages the blood sugar levels of both fasting and post prandial states^{9,10}.

Diabetes affects both the vascular system and the myocardium of the heart. CAD is the most common cardiac manifestation in diabetic patients, followed by dilated cardiomyopathy and autonomic cardiovascular neuropathy.

Women who have diabetes loose their protection against coronary artery disease¹¹. Cardio vascular disease accounted for 65% of death in women with diabetes in a western based population study¹².

In OASIS study¹³ it was found that diabetes increased the mortality risk by 57.6% and in the FINISH study it was observed that men with diabetes had 28day mortality risk of 58%¹⁴. The relation between diabetes and CAD was further supported by the INTERHEART study¹⁵

Presenting high blood sugar levels has been considered as an independent risk factor of death in patients with or without diabetes¹⁶. High blood sugar levels at admission can either be diabetes or due to stress hyperglycemia or impaired glucose tolerance.

Hence it is important to study the spectrum of clinical presentation and the patterns of involvement of CAD in both diabetics and non-diabetics. It has been proved in many studies that diabetes increases the mortality risk in CAD.

The varying patterns of involvement of CAD in patients in relation to their glycemic status was studied in detail in a point to analyse any profound differences existed in the prediabetic group. The severity of coronary artery involvement in diabetics, prediabetics and non-diabetics were assessed quantitatively by means of GENSINI score¹⁷ [Angiographic severity score] in our study. We have analysed the changes in patterns of CAD in patients according to their glycaemic status. The quantitative comparison of CAD and its influence by blood sugar levels and glycaemic status has been analysed in this study.

Prevalence of Diabetes in INDIA and its burden

As of in 2011 International Diabetes federation (IDF) stated that 61.3 million Indians were suffering from diabetes. They have speculated a further rise to 101.2 million by the year 2030. The prevalence of diabetes in Indian studies is 7.3% vs 3.1% in urban and rural areas respectively¹⁸. In Indian reports, the prevalence of cardiovascular disease among diabetics is 30.4 per cent¹⁹ and 39.1 per cent²⁰ in national and international prospective registries respectively.

In India, CAD incidence is increasing over the last 30 years, while a declining trend has been noted in the western population²¹. Reports have shown that Asian Indians have 3-4 times higher risk of CAD than white Americans, 20 times higher than Japanese and 6 times higher than chinese^{22,23}

The reported incidence of CAD among people less than 40 years of age is around 12-16%. Of which 52% of CVD deaths occurred in people below 50 years, and 25% of acute myocardial infarction (AMI) occurred in people less than 40 years in INDIA²¹.

AIM OF THE STUDY

Primary Aim:

1. To assess the severity of CAD by coronary angiography in pre-diabetic, diabetic and non-diabetic patients presenting with acute coronary syndrome
2. To assess the correlation between glycemic control and severity of CAD

Secondary Aim:

1. To assess the relationship between the duration of diabetes and severity of CAD in diabetic patients

REVIEW OF LITERATURE

History of diabetes

Egyptian physician Hesy-Ra first mentioned in the manuscripts of Ebers Papyrus in 1500 BC mentioning it as ‘too great emptying of urine’²⁴. In Indian medicine, Charak and Sushruta, had written between 400 and 500 BC. Urine in diabetes was described by Charak as ‘madhumeha’ or *honey urine* noting that the urine would attract ants. They were also the first to separate Type 1 and Type 2 diabetes as separate conditions referring Type 2 diabetes to be more associated with overweight²⁵. Greek physician Aretaeus of Cappadocia gave a brief description of diabetes, in 120 CE, which he mentions it as ‘the melting down of flesh and limbs into urine’.

It was in 1889, Joseph Vonmering and Oskar Minkowski were the first to discover an association between pancreas and diabetes. They experimented in dogs and found that after removing their pancreas, they developed symptoms and signs of diabetes after which they died shortly²⁶. Paul Langerhans discovered the islets of Langerhans in 1869, which were the chief cells of regulating glucose metabolism in the body²⁷.

Banting, Best, and colleagues with chemist Collip with their experiments led to the invention of insulin preparation and their first usage in 1922²⁸.

In 1923, Nobel prize was awarded to Banting and Macleod in the field of Physiology Medicine. The United Nations announced November 14 as ‘The World Diabetes Day’ in honour of Frederick Banting’s birthday.

History of coronary artery disease

William Heberdan an English physician in 1768 announced his observations about the relationship of coronary vascular anatomy and ischemic heart disease. For a long time in history even up to 19th century the disease was called the 'Heberdan's disease'

Ludwig Hekben in 1879, by his studies concluded that myocardial infarction was due to coronary obstruction secondary to thrombosis.

In 1929, Werner Forssmann was the first to undergo self human catheterization, which he did on himself. Forssmann, a urologist while working on his experiments introduced a catheter into his jugular vein, and he immediately took an X-ray and found it to be inside the right atrium^{29,30}.

According to Cournand, the first catheterization was done by Claude Bernard in 1844 in a horse³¹. André Cournand together with Dickinson Richards successfully repeated in 1941 the trial of Forssmann. With their repeated attempts and experiments they were finally able to push the catheter in to right ventricle and take angiograms. Finally in 1950's the forthcoming experiments by them were a milestone in history towards the present day cardiac interventions³².

Coronary arteriography and ventriculography became the gold standard for studying the vessel anatomy and left ventricular pump function. They gave valuable information and made way for surgical treatment for coronary revascularizations. The initial attempts and repeated experiments by Dotter and Judkins, Andreas Gruntzig led to the advances in invasive cardiology.

DIABETES AND CAD

Diabetes mellitus is a chronic illness characterized by hyperglycemia resulting due to defects in insulin secretions, insulin actions or both. The chronic state of hyperglycemia results in both microvascular and macrovascular complications which causes damage to various organs, especially the eyes, kidneys, nerves, heart and blood vessels.

Classification of Diabetes mellitus

Classification of diabetes according to **American diabetes association (2011)**³³ include four clinical classes:

Type 1 diabetes	characterized by Beta cell destruction, resulting in absolute insulin deficiency
Type 2 diabetes	Characterized by defects in the secretion of insulin and insulin resistance
specific types	genetic defects in β cell function, diseases of the exocrine pancreas (such as cystic fibrosis), genetic defects in insulin action, and drug or chemical-induced (such as in the treatment of HIV/AIDS or after organ transplantation)
Gestational diabetes mellitus (GDM)	diabetes during pregnancy

Criteria for the diagnosis of diabetes³³

HbA1C > 6.5%.

or

FBS>126 mg/dl . (Fasting is defined as no food / caloric intake for at least 8 h)

or

2 hr plasma sugar >200 mg/dl during an OGTT.

or

In a patient with random plasma glucose >200 mg/dl and classic symptoms of hyperglycemia
or hyperglycemic crisis.

Categories of increased risk for diabetes (prediabetes)³³

FBS 100–125 mg/dl (IFG)

or

2-h plasma glucose in the 75-g OGTT : 140–199 mg/dl (IGT)

or

HbA1C 5.7–6.4%

Definition of Myocardial Infarction (ESC/ACCF/AHA/WHF Expert Consensus Document)³⁴

Acute myocardial infarction is defined as evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischemia.

Any one of the following criteria meets the diagnosis for MI:

Detection of a fall or rise in cardiac enzymes with at least one of the following:

- Symptoms of myocardial ischemia.
- New onset or presumed new significant ST-segment / T wave changes or new left bundle branch block (LBBB).
- Development of pathological Q waves in the ECG.
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
- Identification of an intracoronary thrombus by angiography or autopsy.

CORONARY ATHEROSCLEROSIS

The process of atherosclerosis starts with the process of adherence of circulating blood monocytes to the endothelium. These monocytes then migrate to the sub-endothelial space, and further gets activated into monocyte-derived macrophages. Low-density lipoprotein (LDL) particles circulating in plasma slowly invades into the endothelium and gets oxidised. The process of lipoprotein oxidation has been observed since childhood. These oxidised lipoprotein particles release cytokines which attract the monocytes^{35,36}. This initial insult to the endothelium triggers an inflammatory response. Monocytes enter the arterial wall from the bloodstream and the platelets get adhered to the area of inflammation. The monocytes differentiate into macrophages, which ingest the oxidized LDL, and turn into specialised cells called the "foam cells"^{37,38}. These foam cells gradually evade and accelerate the inflammatory process. There is proliferation of smooth muscle cells. They slowly migrate from the tunica media into the tunica intima in response to cytokines. This causes the formation of a fibrous capsule covering the fatty streak. Enzymatic degradation occurs by

time and erodes the fibrous membrane beneath the endothelium. As time passes, the endothelial cap separating the plaque and blood flow becomes thin and fragile. The mechanical stress on the arteries causes these thin membrane to rupture and causes spilling of these plaque contents into blood stream triggering thrombosis³⁹. Blood clotting system gets activated and forms a clot around this site of plaque rupture. This results in unstable angina.

Figure 1: Pathogenesis of atherosclerosis

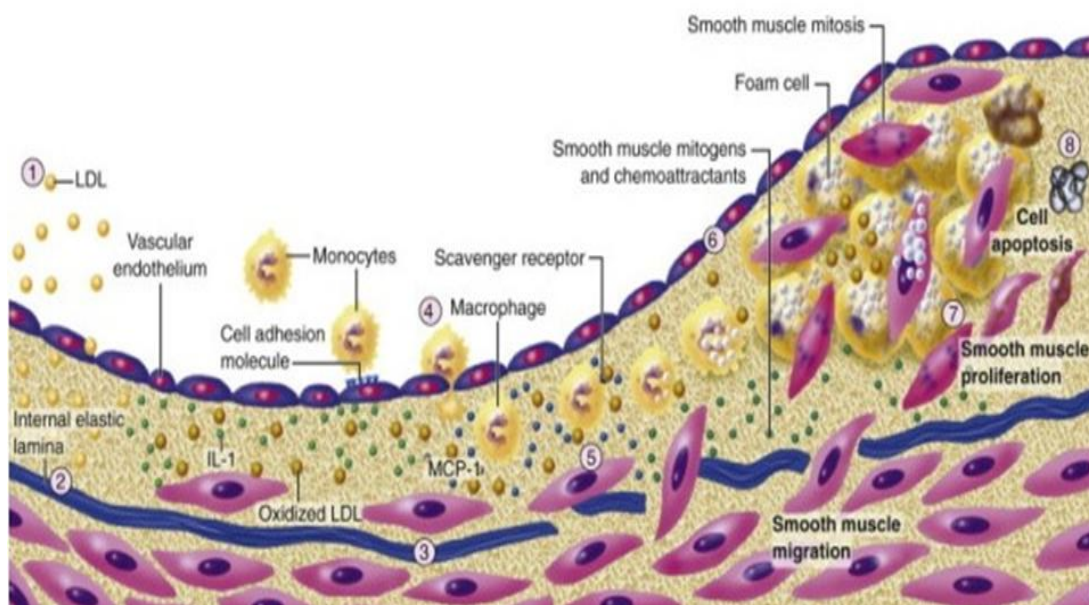


Figure 2: Plaque formation

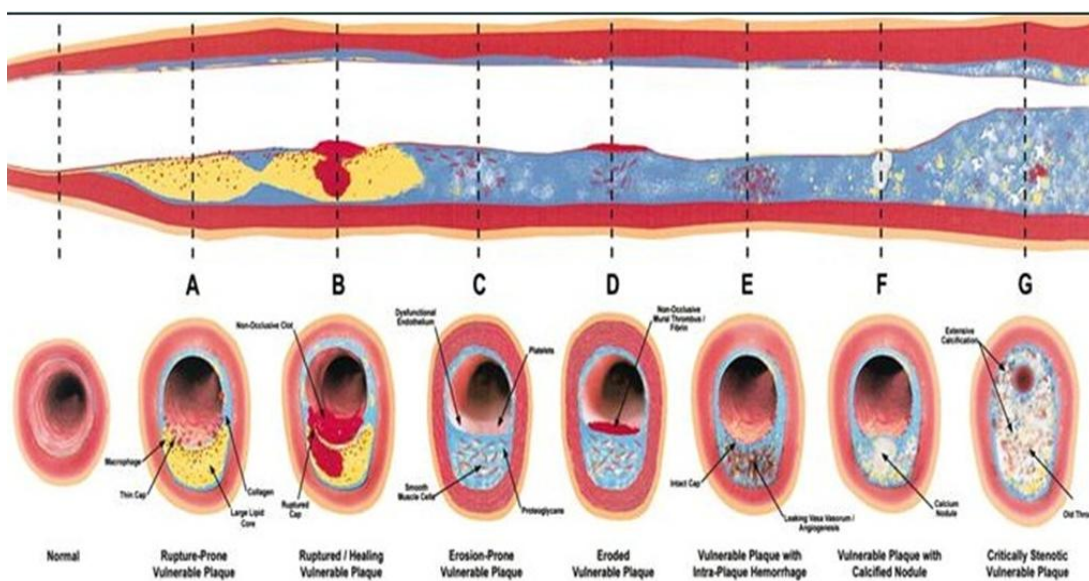
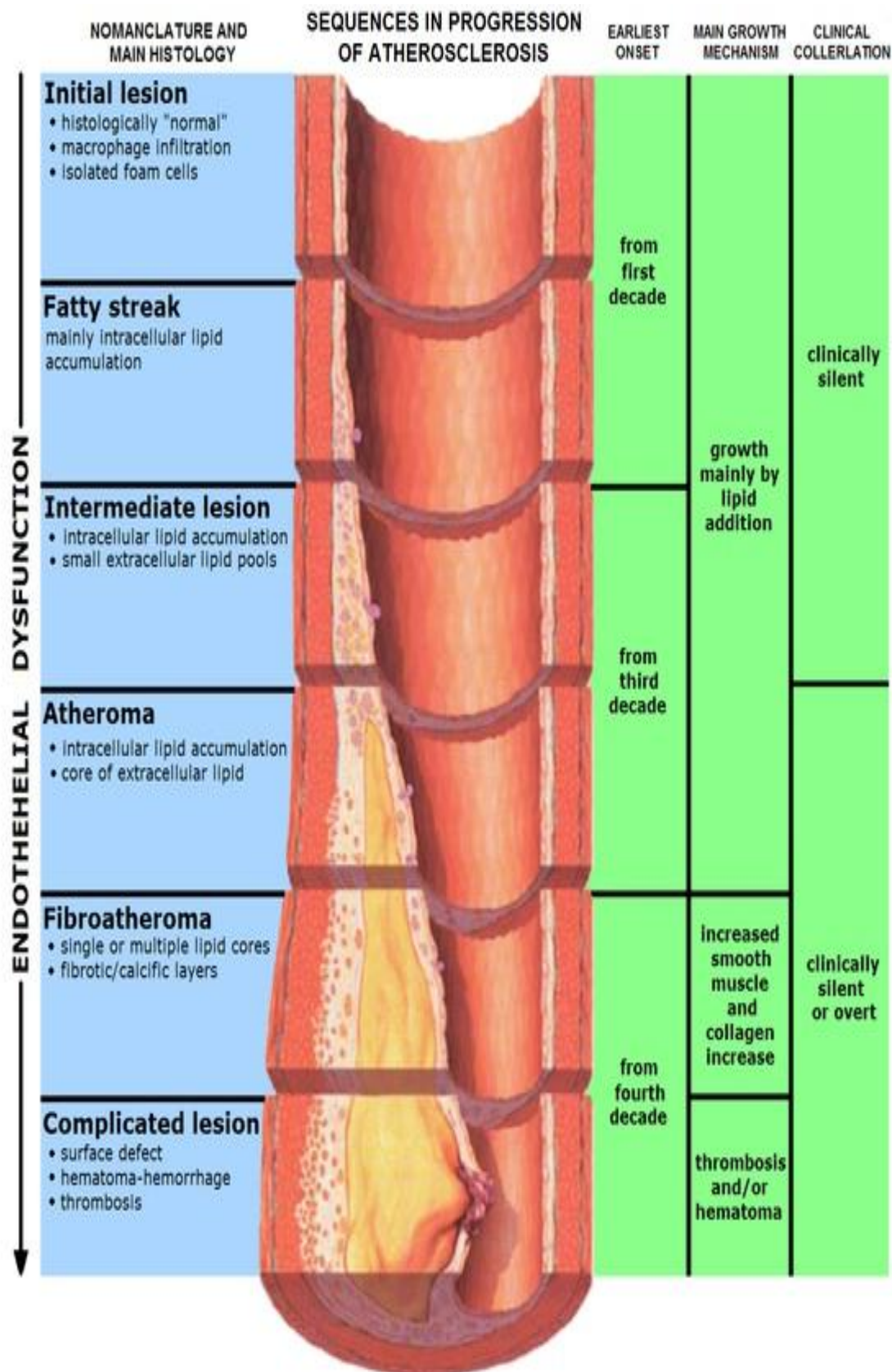


Figure 3: Evolution of Atherosclerosis



PREDIABETES

Prediabetes (IFG and or IGT) is a stage in the natural history of diabetes. It represents a clinical state for (1) progression to diabetes mellitus (increased risk for diabetes) and (2) an increase in cardiovascular and possibly microvascular complications.

The period of transition from prediabetes to diabetes is usually many years, but also can be rapid^{40,41}. Many studies and researches have estimated that upto 70% of individuals progress to diabetes⁴². The incidence is high in people with combined IFG and IGT rather than isolated IFG or IGT. People with IFG or IGT have 5-10% per year risk of developing diabetes, whereas people with normoglycemia have 0.7% per year risk of developing diabetes⁴².

Many studies have shown that prediabetes (IGT and IFG) is associated with an increased risk for cardiovascular problems^{43,44,45}. Usually they are associated with other cardiovascular risk factors, such as obesity particularly abdominal or visceral obesity, dyslipidemia and hypertension. There is increasing evidence that cardiovascular risk increases continually with increasing FBS levels. Prediabetes is associated with the development of microangiopathy. It was observed in 7.9% of patients with IGT to have diabetic retinopathy in Diabetes prevention program⁴⁶.

Metabolic Syndrome and CVD

Metabolic syndrome increases the risk of CAD. In Framingham study⁴⁸, metabolic syndrome alone predicted $\approx 25\%$ of all new-onset CVD. Metabolic syndrome chiefly comprises of glucose intolerance, obesity, Hypertension, micro albuminuria, hyperinsulinemia with hypertriglyceridemia, low HDL levels, and hypofibrinolysis. The pathogenesis is mainly attributed to insulin resistance. Insulin resistance promotes the progression of atherosclerosis even before it develops into frank diabetes. Hence insulin resistance is considered as an independent risk factor for atherosclerosis.

According to ATP III guidelines⁴⁷ presence of any 3 of 5 risk factors listed below is considered as metabolic syndrome

Risk Factor	Defining Level
Abdominal obesity, given as waist circumference	
Men	>102 cm (>40 in)
Women	>88 cm (>35 in)
Triglycerides	≥ 150 mg/dL
HDL cholesterol	
Men	<40 mg/dL
Women	<50 mg/dL
Blood pressure	$\geq 130/\geq 85$ mm Hg

At least three of the above ATP III criteria fit into metabolic syndrome. Atherosclerosis in type 2 diabetes mellitus is due to insulin resistance and hyperinsulinemia. Both of these mechanisms sets early in prediabetics, which precedes the onset of diabetes and impaired glucose tolerance by many years.

IFG is associated with hepatic insulin resistance, which results in fasting hyperglycemia, whereas IGT is predominantly associated with muscle insulin resistance. Most of the patients with prediabetes and metabolic syndrome are obese. There is an increase in adipose tissue which causes an increase in the levels of circulating free fatty acids (FFA) and adipokines⁴⁹. These adipokines produce a proinflammatory and a prothrombotic state. The increase in FFA induces insulin resistance in muscles. In a chronic state they lead to lipotoxicity and produces beta cell dysfunction⁵⁰. Elevated levels of FFA leads to increased hepatic glucose secretion and worsening hyperglycemia. They are also responsible for an increase in triglyceride levels and lowers HDL levels. Insulin resistance gradually presets a proinflammatory state and predisposes to cardiovascular disease⁵¹.

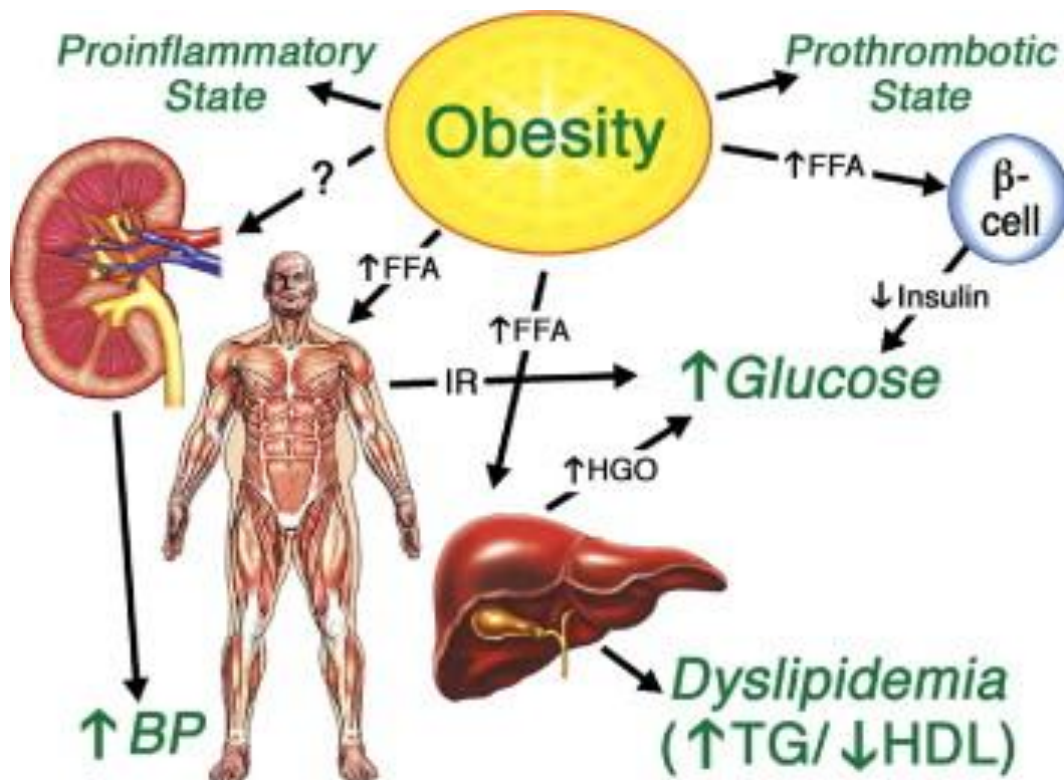
Pathogenesis of CAD in Prediabetes⁵²:

The central process in the pathogenesis of prediabetes is the central obesity (measured by increased W:H ratio), which produces a state of insulin resistance and consequent hyperinsulinaemia. Because of the resistance to action of insulin there is a decreased cellular glucose uptake coupled with hepatic glucose production, which gets combined with β -cell exhaustion and produces a state of hyperglycemia.

Mechanisms: Hyper insulinaemia causes:

1. Increased renal Na⁺/water re-absorption.
2. Over activity of sympathetic nervous system.
3. Increased Na⁺-H⁺ pump activity.
4. Decreased Na⁺ -K⁺ ATPase activity.
5. Increased Ca²⁺ concentration (calcium-cytosolic) in cells.
6. Proliferation of arterial smooth muscle cells.

Figure 4: Factors confounding in metabolic syndrome

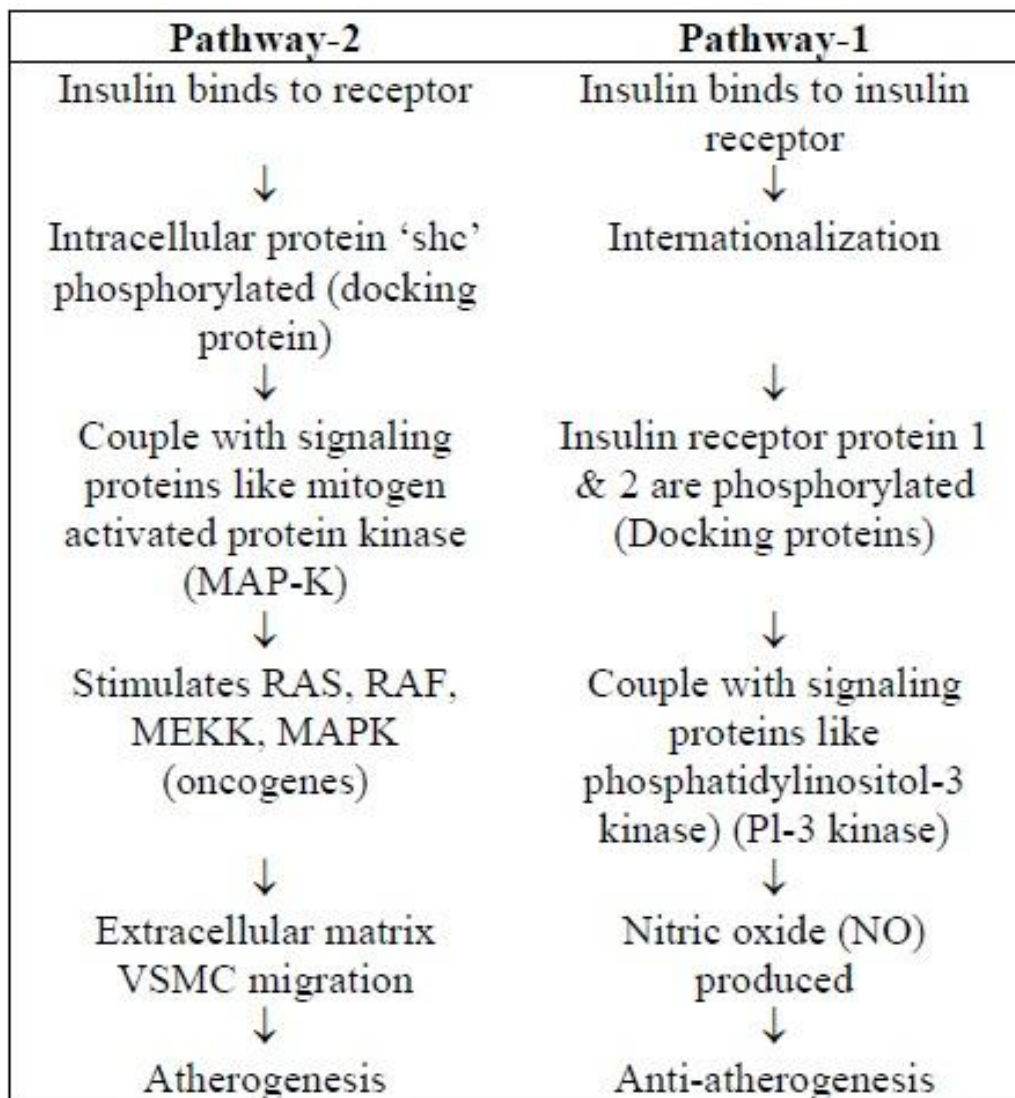


Role of Insulin on Vascular Endothelium, in normal individuals:

Insulin has both anti-atherogenic and atherogenic properties on vascular endothelium.

Anti-atherogenic	Atherogenic
Insulin stimulates the production of nitric oxide from vascular endothelium which causes	
Vasodilatation.	Vascular cell growth
Prevents platelet aggregation.	
Prevents growth of vascular smooth muscle cells (VSMC)	Synthesis of extracellular Proteins and type IV collagen.
Prevents migration and proliferation of VSMC.	Modest effect on growth of VSMC and atherogenesis.
Prevents formation of foam cells.	Stimulation of growth factors.
Inhibits expression of vascular adhesion molecules and intercellular adhesion molecules.	Enhanced LDL receptor activity and cholesterol synthesis.
Inhibits proinflammatory INF	
Inhibits production of monocytes chemo attraction protein-1 (MCP-1)	

Mechanisms by which insulin produces Atherogenesis and anti-atherogenesis:



Insulin Resistance⁵³:

Insulin resistance is a metabolic state in which a normal concentration of insulin produces a less than normal biologic response. Insulin resistance results from abnormalities at any point in the sequence of insulin synthesis and secretion, to its transport and action on target tissue level i.e., at receptor, pre-receptor, post receptor levels. With respect to diabetes and CAD, both pre-receptor and post-receptor mechanisms are important.

Insulin resistance sets in hyperinsulinemia which has been implicated for an increased incidence of coronary artery disease⁵⁴⁻⁵⁷. Insulin causes vasodilation through secretion of endothelial nitric oxide synthase. These mechanisms are affected in insulin resistance.

Endothelial dysfunction

Endothelium has unique characteristics to maintain blood in a liquid state during circulation. These properties of endothelium are rendered by the expression of heparin sulphate and thrombomodulin on the surface of endothelium that inactivates the thrombin activation⁵⁸. Endothelium also possesses fibrinolytic properties by production of urokinase type plasminogen activators, thus helps in maintaining an anti-thrombotic and anti-coagulant properties⁵⁹. It also releases nitric oxide, prostaglandins, endothelin, angiotensin II. Nitric oxide produces vasodilation, which also reduces the inflammation caused by the adhesion of leucocytes and its migration thereby decreasing the smooth muscle cell proliferation⁶⁰⁻⁶². All these protective mechanisms against atherogenesis are lost in Diabetes mellitus, increasing their relative cardiovascular risk. These changes are even apparent in patients with insulin resistance well before their progression into overt hyperglycemia.

Hyperglycaemia

In atherogenesis there is a migration of monocytes and T-lymphocytes into the intima, producing cytokines. These monocytes ingest the LDL and form the Foam cells, which are the early precursors of atherosclerosis^{63,64}. Hyperglycemia augments these pathological changes such as increased oxidative stress, non-enzymatic glycosylation of proteins and lipids, protein C activation and activation of transcription factors⁶⁵.

Dyslipidemia

In diabetes, hyperglycemia is associated with high triglyceride levels, increased atherogenic low density lipoproteins, and lower high density lipoproteins. Moreover hyperglycemia promotes the glycosylation of LDL, thereby promoting the inflammatory action of macrophages to form foam cells. All these factors contribute to an increased risk of atherosclerosis in diabetes⁶⁶. There is an increased hepatic free fatty acid concentration due to increased efflux from adipose tissues and insulin mediated skeletal muscle uptake, thereby liver increases VLDL production and cholesterol ester synthesis⁶⁷. Overproduction plus decreased clearance of triglyceride rich lipoproteins due to the reduced action of lipoprotein lipase also leads to hypertriglyceridemia⁶⁸. The increase in LDL particles are due to increased VLDL secretion and abnormal triglyceride and cholesterol transfer between VLDL and LDL^{69,70}.

Inflammation

Diabetes is associated with up regulation of C-reactive protein, increased expression of VCAMs, Interleukins and activated protein C. All of which have been attributed to cause adverse cardiac outcomes⁷¹⁻⁷⁴. Elevated C-reactive protein and VCAM levels are independently shown to be associated with poorer outcomes.

Inflammation	<ul style="list-style-type: none">↑ IL-1β, IL-6, CD36, MCP-1↑ ICAMs, VCAMs, and selectins↑ Activity of protein kinase C↑ AGEs and AGE/RAGE interactions
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Prothrombotic state and platelet function

There is an increased expressions of factor VII, Factor VIII, Von willebrand factor, plasminogen inhibitor in diabetic patients. There is also a profound alteration in the platelet function such as a) increased expression of glycoprotein IIb/IIIa, b) decreased Nitric oxide production c) decreased prostacyclin production and d) increased production of fibrinogen. All of which causes a procoagulant state in diabetic individuals⁷⁵.

Perturbations of platelet structure and function associated with diabetes⁷⁵

Reduced membrane fluidity

Altered Ca^{2+} and Mg^{2+} homeostasis

Increased arachidonic acid metabolism

Increased thromboxane A_2 synthesis

Decreased nitric oxide and prostacyclin production

Decreased antioxidant levels

Increased expression of activation-dependent adhesion molecules

(e.g., glycoprotein IIb/IIIa, P-selectin)

Increased platelet microparticle formation

Increased platelet turnover

Family history

The familial trend in the occurrence of CAD and its genetic basis is found in many numbers of genes and are yet expanding in the field of research. Many studies including the 'Framingham study' have found a significant association between family history and CAD and have now been considered as a separate independent risk factor for CAD⁷⁶⁻⁷⁹. Nearly more than 50% of patients who present with premature CAD are found to have genetically linked lipoprotein disorder⁸⁰. The presence of additional risk factor such as smoking, sedentary lifestyle, obesity and dyslipidemia adds up to the relative risk of CAD in people with a positive family history of CAD. In a study in Finland, presence of family history of CAD was found to be associated with increased mortality risk.

EFFECTS OF HYPERGLYCEMIA IN ACUTE MYOCARDIAL INFARCTION

Patients with AMI with hyperglycemia, have increased risk of adverse outcomes in both diabetic and non-diabetic patients⁸⁰. In acute MI, hyperglycemia was found to be associated with worse clinical outcomes. Presence of hyperglycemia resulted in higher incidences of congestive heart failure, cardiogenic shock and death in patients with ACS. A number of mechanisms have been entitled for this increased adverse outcomes in patients with hyperglycemia, of particular interests include

1. Endothelial Dysfunction⁸¹⁻⁸⁸

The possible accepted mechanism for hyperglycemia and poorer cardiovascular outcomes was the effect of acute hyperglycemia on the vascular endothelium. During situations of stress, the endothelium is subjected to various harmful effects such as dysfunction, dysregulation and failure. The resulting endothelial dysfunction ultimately leads

to altered angiogenesis, altered cell permeability, thrombosis and inflammation. Ultimately resulting in a loss of normal endothelial function.

Increased endothelial adhesiveness and enhanced haemostasis are both thought to contribute to atheroma formation. Endothelial vasomotor dysfunction is another abnormality that precedes the development of overt atheromatous disease. Endothelial vasomotor dysfunction presumably reflects wider endothelial damage and it is a powerful predictor of atherogenesis and MI.

2. Impaired collateral formation⁸⁹⁻⁹¹

Collateral development is a cardio protective mechanism in the normal vascular endothelium in response to myocardial ischaemia. The development of collateral vessels was found to be much more impaired in patients with diabetes. In hyperglycemia there is a significant rise in the levels of reactive oxygen species (ROS) in the arteriolar endothelium. This along with added endothelial dysfunction, decreased production of Nitric oxide synthase and reduced production of NO resulted in an impairment of vasodilation and permeability. Thus altering the formation of collaterals. This resulted in an impaired initial vasodilation and permeability, impairing the formation of collateral vessels. Thus impairing the initiation of arteriogenesis and causes a negative effect on the remodelling of collateralization.

3. Increased microvascular dysfunction after reperfusion (hyperglycemia and the No-reflow phenomenon)⁹²⁻⁹⁸

The no-reflow phenomenon is one of the major factors determining the prognostic and clinical outcome of MI. This has been observed in up to 30% of patients with ACS, even after a successful PCI. This results in a large area of myocardial necrosis, poorer recovery and more post MI related complications.

In a study by Iwakura et al, it was found that hyperglycemia was a major factor predicting the occurrence of no-reflow. The large infarcts caused more release of catecholamines, which alters the free fatty acid and glucose metabolism. Acute hyperglycemia also resulted in an increase in levels of P selectin, which further increases the plugging of leucocytes into the capillaries. This clumping or trapping of leucocytes in coronary capillaries and venules early after coronary reperfusion are much more frequently observed in the diabetic heart than in the non-diabetic heart. The plugging of leucocytes in the microcirculation might further add up to the no-reflow phenomenon. Acute hyperglycemia may also augment thrombus formation.

4. Location of lesions

Proximal segment and ostial segment lesions are more commonly observed in patients with Diabetes. These lesions are associated with a lower risk of procedural success and a higher rate of major adverse cardiac events even after PCI. Non-insulin dependent diabetes mellitus was observed to have more left main coronary artery disease⁹⁹.

FACTORS ALTERING THE SEVERITY OF CAD IN DIABETES MELLITUS

Sex

In diabetes, women lose their cardio protection. Increased severity of disease in diabetic women has been noted in various studies¹⁰⁰⁻¹⁰². Women with diabetes had 4 times more chance of developing cardiac failure (16% vs 3.8%) when compared to non-diabetic women.

Possible mechanisms¹⁰³ quoted for this increased mortality risk in female sex include

- a) Low HDL in diabetic women
- b) Insulin androgen interaction
- c) Poorer collateral circulation
- d) Diabetic cardiomyopathy has been noted more common in women

Duration of diabetes and severity of CAD

Extent of coronary artery disease in terms of stenosis percentage has been studied in several studies with respect to duration of diabetes. Many studies have shown that the severity of CAD is increased in individuals with longer duration of diabetes¹⁰⁴⁻¹⁰⁸. The proposed mechanism being diabetic individuals are being exposed to the risk factors even before the establishment of clinical diabetes.

Lipids

Hypercholesterolaemia was considered as a risk factor in addition to CAD in a study of 204 patients in japan¹⁰⁹. Lipoprotein(a) concentration and triglyceride rich lipoprotein particles were considered as confounding factors affecting the severity of CAD in patients with diabetes mellitus¹¹⁰.

METHODOLOGY

SOURCE OF DATA:

This study was undertaken in PSG hospitals, affiliated to PSG institute of medical sciences and research, Coimbatore, Tamil Nadu during the study period between august 2011 to December 2012. A total of 412 patients admitted with acute coronary syndrome in the study period who subsequently underwent coronary angiogram were included in the study.

METHODOLOGY:

All patients were evaluated in detail as per their symptomatology, a detailed physical examination, routine blood investigations with blood sugar estimation and HbA1c, ECG and ECHO were done at admission. They were categorized into non-diabetics, diabetics and pre-diabetics according to their HbA1c levels. Diabetic patients were subcategorized according to their hbA1c levels (good control = <6.5, sub optimal control = 6.5-8.9, poor control = >9). Those patients in pre-diabetic range were evaluated with FBS, PPBS, subjected to oral GTT if necessary and subcategorized into impaired fasting glucose, impaired glucose tolerance or both. All patients were medically stabilised and subjected to coronary angiogram as an when it was indicated. The angiographic severity of the coronary artery disease was assessed using the Gensini score. Proper consent was obtained from individuals enrolled into the study. Ethical clearance was obtained as per institution norms. Statistical analysis was assessed using Mean \pm standard deviation, Pearson's correlation coefficient, Chi square test and ANOVA tests using SPSS software.

Inclusion Criteria:

- Patients admitted with acute coronary syndrome (STEMI, NSTEMI , Marker positive unstable angina) who underwent invasive coronary angiogram were included in the study

Exclusion Criteria:

- Patients with known ischemic heart disease and proven CAD.
- Patients with anaemia, renal failure or other known systemic illness.

PROTOCOL OF THE STUDY

Acute coronary syndrome

ENTRY POINT

DIABETIC CAD

- Known diabetic•

NON-DIABETIC CAD

Not a known diabetic



HISTORY

- Typical/ atypical presentation of CAD (chest pain, dyspnea, sweating, abdominal pain, syncope)
- Presence / absence of diabetes
- Duration and control of diabetes
- Risk factors (Smoking / Family history of IHD/ hypertension)
- History of diabetic/ hypertensive complications
- Other significant history

CLINICAL EXAMINATION

- Stigmata of hypercholesterolemia
- Vital signs
- Pulse (look for rhythm disturbance)
- BP (look for hypertension/ hypotension)
- Cardiac status by Killip classification
- ECG (Type of CAD/ rhythm disturbance)

PATIENT STABILIZATION



INVESTIGATIONS

- Routine blood and urine
 - RBS, FBS & PPBS
 - HbA1c
 - Blood urea, S.creatinine, Urine –Microalbuminuria, Assess nephropathy (if present)
 - Lipid profile
 - Look for dyslipidemia and risk category
 - Fundus examination Diabetic/ hypertensive changes
- ↓
- Cardiac enzymes : Trop T

ESTABLISH DIAGNOSIS

Type of CAD:

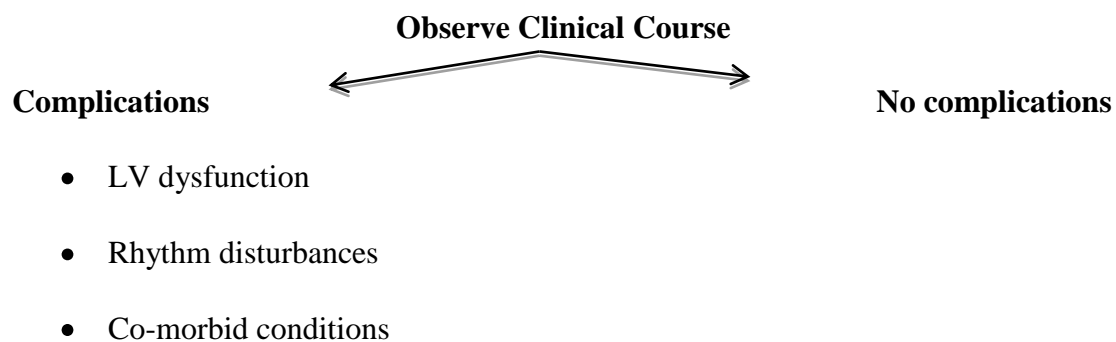
- STEMI AW / IW / IW + RV, NSTEMI, Unstable angina

Echocardiography:

- Regional wall motion abnormality
- Systolic dysfunction
- Diastolic dysfunction

Angiography:

- Gensini score – categorisation



DEFINITIONS:

Coronary Artery Disease (A.M.I) is defined in the presence of minimum 2 of following³⁴.

1. Chest pain suggestive of Cardiac Origin.

[Retrosternal squeezing. Radiating, increases with exertion, not relieved by rest and nitrates and associated with sweating.]

2. Troponin T positive.

3. Electrocardiographic changes.

This consists of ≥ 1 of the following.

- a) ST segment elevation of more than 2mm from J Point in 2 related electric fields with typical evolutionary changes.
- b) Presence of new pathologic Q waves in 2 related electric fields. [for Q-M.I.]
- c) Non Q-M.I. with ST depression and T inversion

DIABETES (ADA CRITERIA)

Criteria for the diagnosis of diabetes³³

HbA1C > 6.5%.

or

FBS >126 mg/dl . (Fasting is defined as no food/ caloric intake for at least 8 h)

or

2-h plasma glucose >200 mg/dl during an OGTT.

or

In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose >200 mg/dl.

Categories of increased risk for diabetes (prediabetes)³³

FBS 100–125 mg/dl (IFG)

or

2-h plasma glucose in the 75-g OGTT: 140–199 mg/dl (IGT)

or

HbA1C 5.7–6.4%

Classification of Hypertension (JNC7)

Blood Pressure classification	SBP mmHg	DBP mmHg
Normal	<120	and <80
Prehypertension	120–139	80–89
Stage 1	140–159	90–99
Stage 2	>160	>100

Classification of Lipid profile

Third Report of the National Cholesterol Education Program (NCEP)

Total Cholesterol (mg/dL)	LDL Cholesterol (mg/dL)	Triglyceride (mg/dL)	Serum HDL Cholesterol (mg/dL)
<200 Desirable	<100 Optimal	<150 normal	<40 Low HDL
200–239 Borderline High	100–129 Near optimal	150–199 borderline high	>60 High HDL
High >240	above optimal 130–159	200–499 high	
	160–189 High	>500 very high	
	>190 Very High		

Severity of CAD

Gensini score was used to assess the severity of CAD¹⁷.

Specific score is used to assess the narrowing of the coronary artery. First the percentage of stenosis is assessed.

Stenosis score	Percentage of occlusion
1	1 – 25
2	26 - 50
4	51 - 75
8	76 - 90
16	91 - 99
32	Complete occlusion

This stenosis score is then multiplied by a factor according to the importance of the coronary artery. The multiplication factor for a left main coronary artery (LMCA) lesion is 5, 2.5 for proximal left anterior descending artery (LAD) and proximal circumflex artery (LCX) lesions, 1.5 for a mid-LAD lesion, and 1 each for distal LAD, mid LCX, distal CX and right coronary artery. The multiplication factor for any other branch is 0.5.

Gensini score = sum of (stenosis score x functional significance score)

Significance score

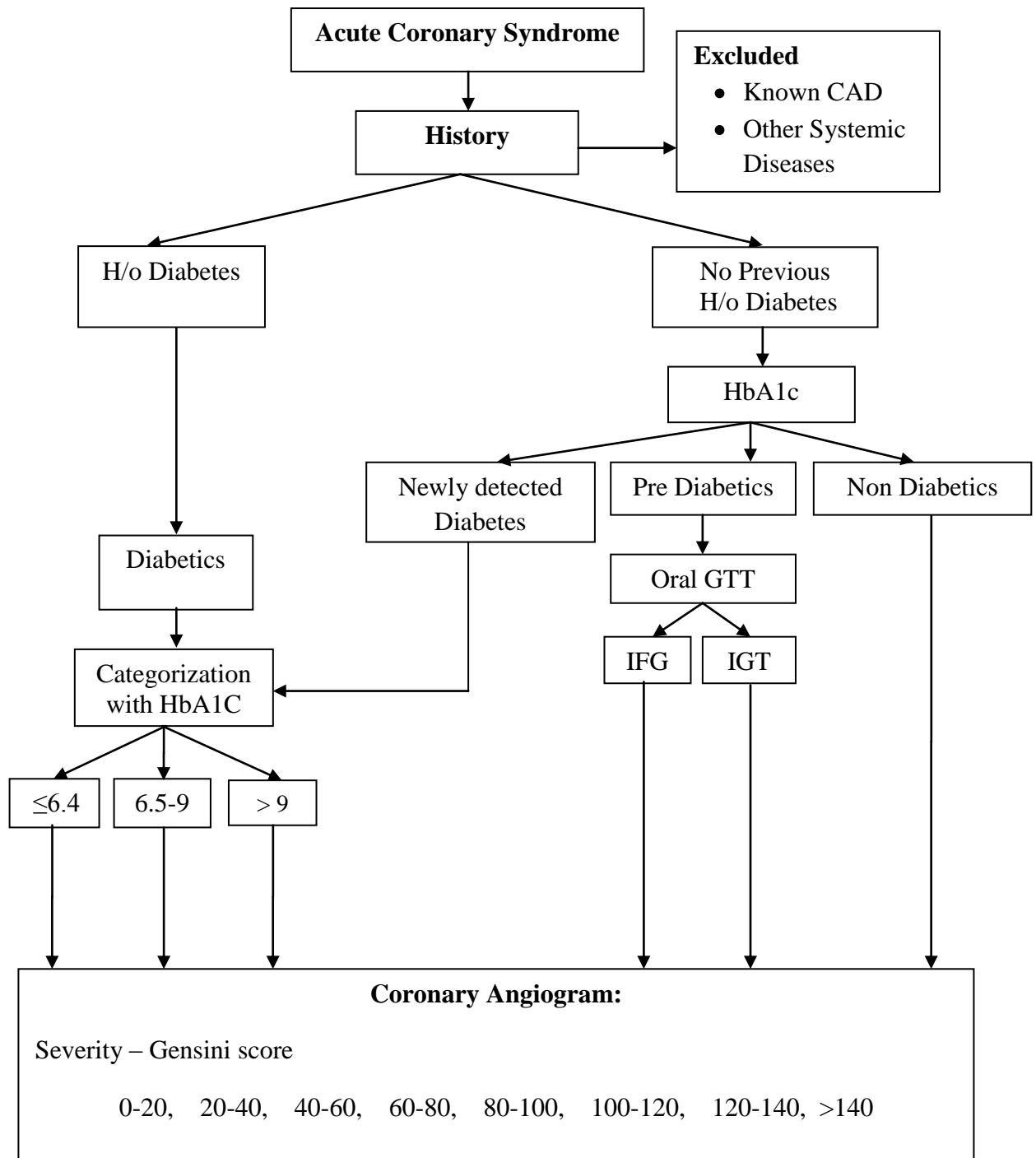
LM : 5	d-LCx : 1
p-LAD : 2.5	OM : 1
m-LAD : 1.5	p-RCA : 1
d-LAD : 1	m-RCA : 1
1 st Dx : 1	d-RCA : 1
2 nd Dx : 0.5	PD : 1
p-LCx : 2.5	PL : 1

Stenosis score

1 : 25%	2: 50%
4 : 75%	8: 90%
16: 99%	32: 100%

LM indicates left main, LAD, left anterior descending; Dx, diagonal; OM, obtuse marginal; RCA, right coronary artery; PD, posterodescending; PL, posteolateral; p-, proximal; m- mid; d- distal

FLOW CHART OF THE STUDY



RESULTS AND OBSERVATION

There were 412 patients with first time ACS in the study population. Of them 213 were Diabetics, 123 were non-diabetics, and 76 were prediabetics

CATEGORISATION IN THE STUDY GROUP

Table 1 Group distribution of study population

Group	Total number of cases	Percentage (%)
Diabetics	213	51.7
Prediabetics	76	18.4
Non diabetics	123	29.9
Total	412	100

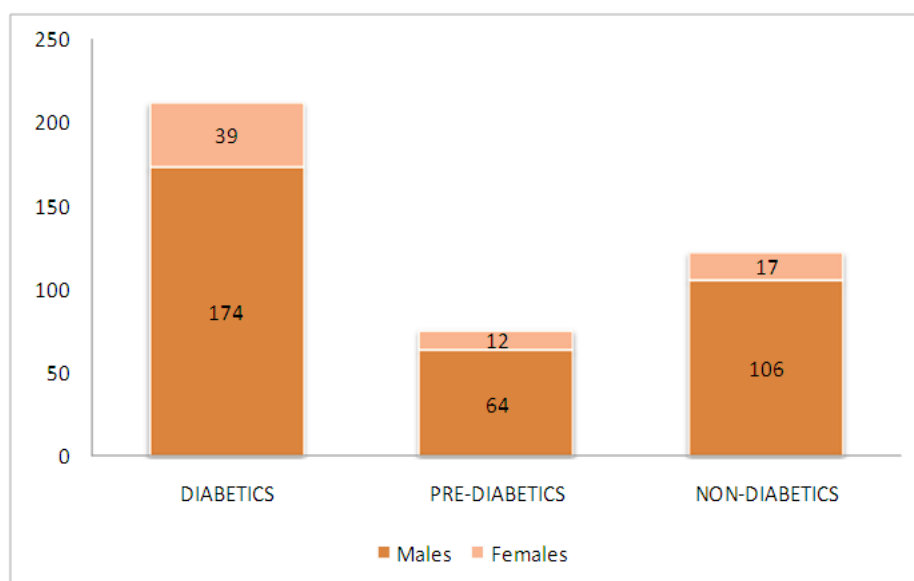
SEX DISTRIBUTION AMONG STUDY GROUPS

Table 2 Sex wise distribution of study group

Group	Male (%)	Female (%)	Male : Female
Diabetics	174 (81.7)	39 (18.3)	4.46:1
Pre-Diabetics	64 (84.2)	12 (9.8)	5.33:1
Non-Diabetics	106 (86.2)	17 (13.8)	6.23:1

There was male predominance in all the study groups, with the highest noted in non-diabetics with 6.23:1

Figure 5: Sex distribution



AGE WISE DISTRIBUTION OF STUDY GROUPS

Table 3 Mean age at presentation

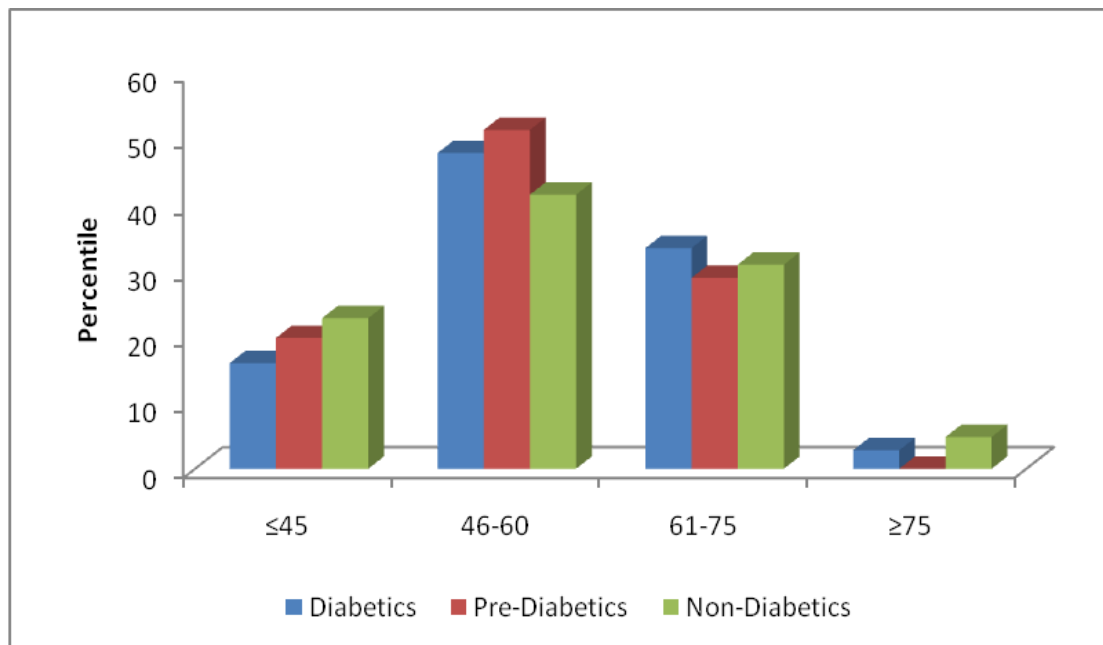
Group	Mean Age
Diabetics	56.3± 10.2
Non diabetics	54.5 ±12.2
Pre diabetics	54.4± 9.3

Mean Age at presentation was higher in diabetics with 56.3 years.

Table 4 Age distribution of the study groups

Age in years	≤45	%	46-60	%	61-75	%	≥75	%	Total
Diabetics	34	16	102	47.8	71	33.4	6	2.8	213
Pre-Diabetics	15	19.8	39	51.3	22	28.9	-	-	76
Non-Diabetics	28	22.8	51	41.5	38	30.9	6	4.8	123

Figure 6: Age distribution of study groups



In all the 3 groups, patients in age group between 46-60 years were mostly affected. There were no patients above 75 years in the prediabetic group. In patients above 61 years most of them were non-diabetics.

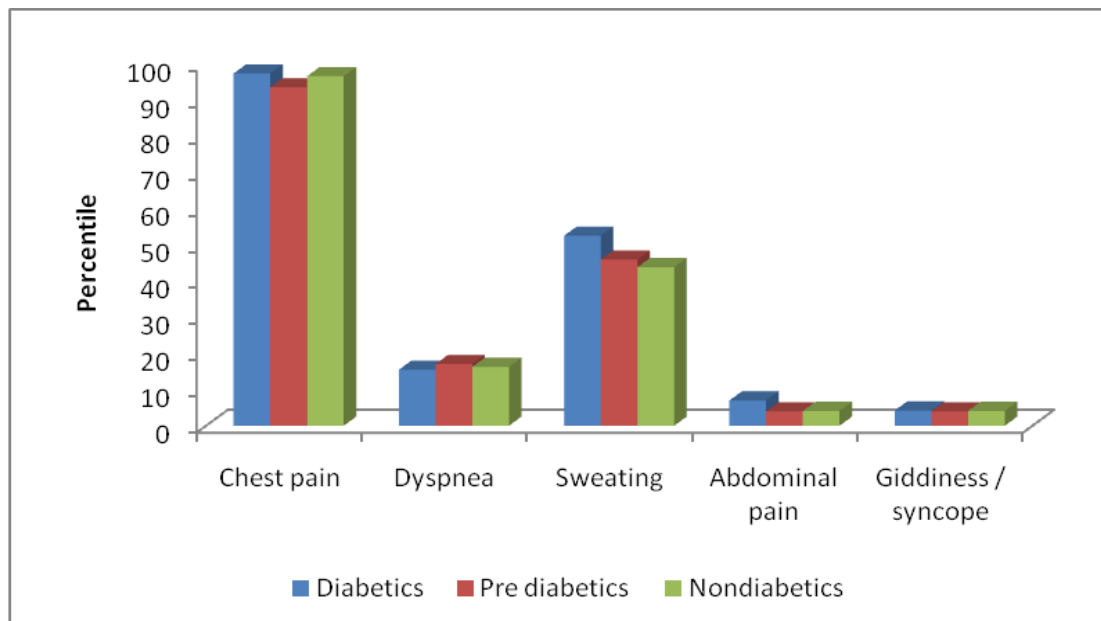
SYMPTOM ANALYSIS OF STUDY GROUPS

Table 5: Symptom analysis of study groups

Symptom	Diabetics		Pre diabetics		Nondiabetics	
	Number	Percentage	Number	Percentage	Number	Percentage
Chest pain	208	97.5	74	93.7	119	96.75
dyspnea	33	15.5	13	17.1	20	16.3
sweating	112	52.6	35	46	54	43.9
Abdominal pain	15	7	3	3.95	5	4.1
Giddiness / syncope	9	4.2	3	4	5	4.1

There were no notable differences in the presenting symptom among the study groups. Most common symptom being chest pain and sweating in all the 3 groups.

Figure 7: Symptom analysis of study groups



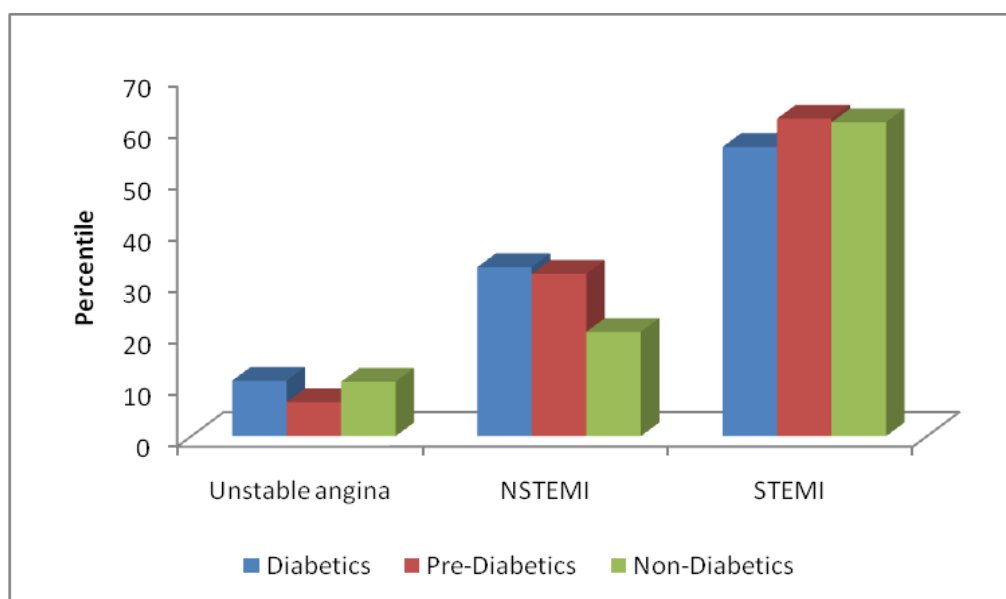
ACS DISTRIBUTION

Table 6: ACS distribution among study groups

Group	Unstable angina	NSTEMI	STEMI	total	Distribution of STEMI cases			
					AW	IW	IW+ RV	LW
Diabetics	23 10.8%	70 32.9%	120 56.3%	213	68	40	9	3
Pre-Diabetics	5 6.6%	24 31.6%	47 61.8%	76	25	19	2	1
Non-Diabetics	13 10.6%	25 20.3%	85 69.1%	123	59	21	4	1
P value 0.059								

Of the ACS distribution, majority of the patients in all the 3 groups presented with ST elevation myocardial infarction. STEMI presentation was particularly higher in Non-diabetics, compared to other groups. Whereas NSTEMI and unstable angina were higher in diabetic group. However it failed to prove statistical significance.

Figure 8: Distribution of ACS among the study groups



RISK FACTORS IN STUDY GROUPS

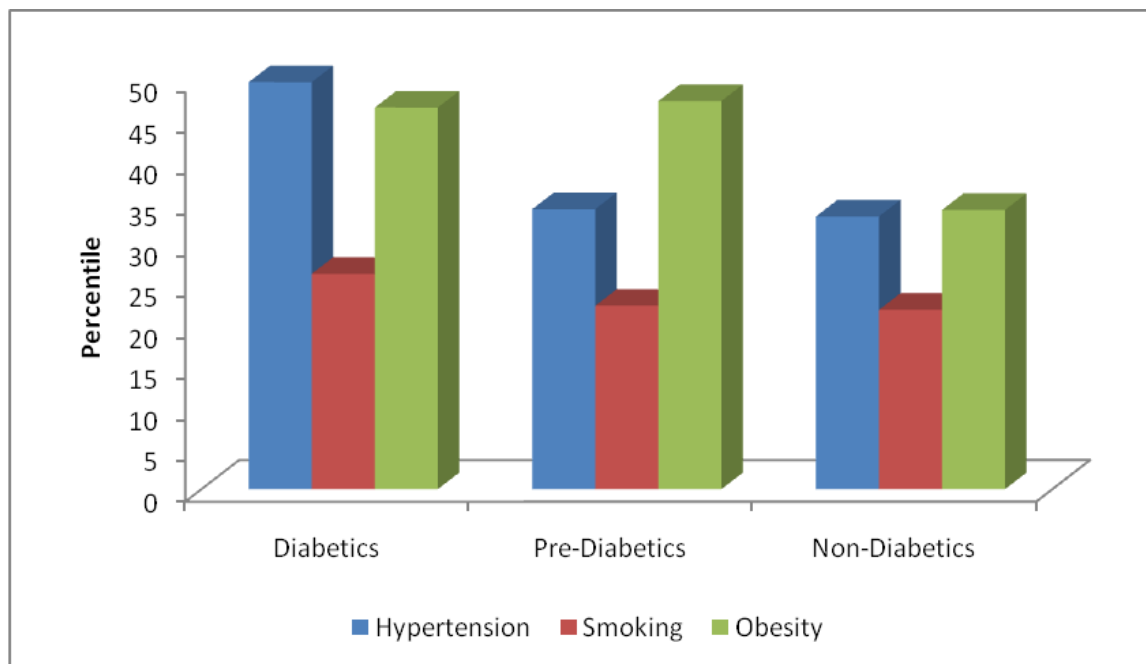
Table 7: Prevalence of risk factors in study groups

Risk factor	Diabetics	Pre-Diabetics	Non-Diabetics
Hypertension	106 (49.7%)	26 (34.2%)	41 (33.3%)
Smoking	56 (26.3%)	17 (22.4%)	27 (21.9%)
Obesity	95 (46.6%)	36 (47.4%)	42 (34.1%)

P value 0.002

Obesity was more prevalent in pre diabetics, followed by diabetics. Hypertension was more prevalent in diabetics. There was no much variation in the prevalence of smoking in the study groups

Figure 9: Risk factors among study groups



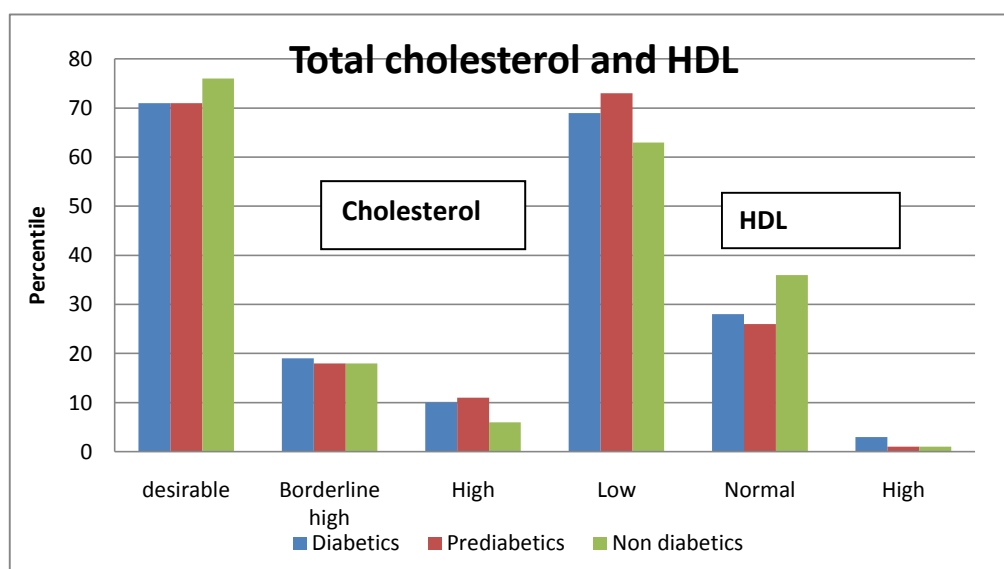
LIPID ABNORMALITIES IN THE STUDY GROUPS

Table 8: Total cholesterol and Triglyceride Levels in Study groups

	Total cholesterol			HDL		
	Desirable (%)	Borderline High (%)	High (%)	Low (%)	Normal (%)	High (%)
Diabetics	150 (71)	40 (19)	21 (10)	148 (69)	59 (28)	6 (3)
Prediabetics	54 (71)	14 (18)	8 (11)	55 (73)	20 (26)	1 (1)
Non Diabetics	94 (76)	22 (18)	7 (6)	78 (63)	44 36)	1 (1)

There was no much observational variation in the levels of total cholesterol in the study groups. Whereas lower levels of HDL cholesterol was noted more in Prediabetics. Very high levels of LDL cholesterol was noted in 9% of prediabetics and 6% of Diabetics.

Figure 10: Total cholesterol and Triglyceride Levels in Study groups

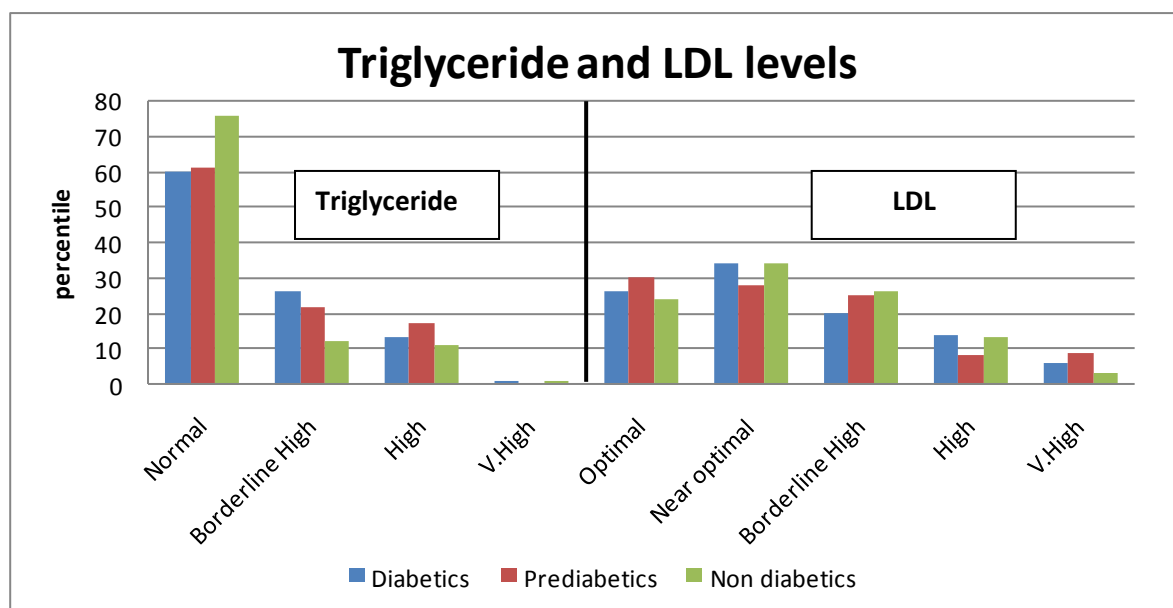


TRIGLYCERIDE AND LDL LEVELS

Table 9: Triglyceride and LDL levels

	Triglyceride				LDL levels				
	Normal	Border line high	High	Very high	Optimal	Near optimal	Border line high	High	Very high
Diabetics	128 (60)	55 (26)	28 (13)	2 (1)	54 (26)	74 (34)	43 (20)	29 (14)	13 (6)
Prediabetics	46 (61)	17 (22)	13 (17)	0 (0)	23 (30)	21 (28)	19 (25)	6 (8)	7 (9)
Non Diabetics	94 (76)	15 (12)	13 (11)	1 (1)	30 (24)	42 (34)	32 (26)	16 (13)	3 (3)

Figure 11: Distribution of Triglyceride and LDL levels



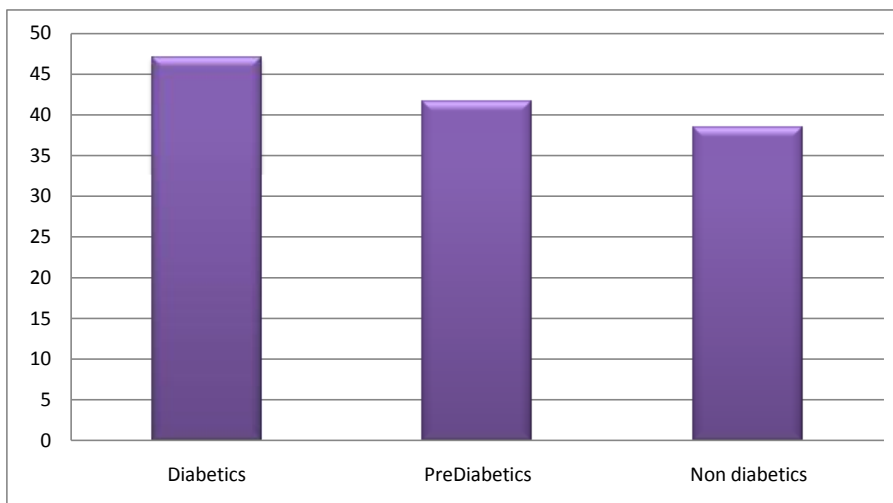
ANGIOGRAPHIC SEVERITY OF CAD IN DIFFERENT GROUPS

Table 10: Angiographic severity of CAD in different groups

Group	Mean HbA1c	Mean Gensini score	P value
Diabetics	8.23±1.88	47.1±31.1	0.049
PreDiabetics	6±0.19	41.5±25.1	
Non diabetics	5.40±0.30	38.45±33.9	

The severity of CAD was higher in Diabetics with a mean gensini score of 47.1, followed by Prediabetics with 41.5 and Non diabetics with comparatively lower involvement of severity of CAD with a mean gensini score of 38.5. (P value 0.049)

Figure 12: Angiographic severity of CAD in different groups



DISTRIBUTION OF GENSINI SCORE IN PREDIABETIC GROUP

Table 11: Distribution of gensini score in prediabetic group

Group	Total patients (% among prediabetics)	Mean gensini score
IFG	57 (75)	44.4±25.3
IGT	58 (76.3)	41.03±25.7
IFG & IGT	43 (56.5)	43.86±26.85

P value 0.12

Around 56% of patients in prediabetic group had both IFG and IGT. There was no much statistically significant difference in severity of CAD among these groups.

DURATION OF DIABETES AND GENSINI SCORE IN THE DIABETIC GROUP

The severity of CAD was higher in patients with diabetes for more than 5 years. There was statistically significant correlation between the duration of diabetes and severity of CAD in diabetics.

Table 12: Duration of diabetes and gensini score in the diabetic group

Duartion	Newly detected	0-1 yrs	1-3yrs	3-5 yrs	5-10yrs	>10yrs
Diabetic group	56	14	33	34	40	36
Mean gensini score	34.5	27.4	26.8	42.5	57.8	85

Correlation coefficient $r=0.69$, $p<0.001$

Figure 13: Duration of diabetes and gensini score in the diabetic group

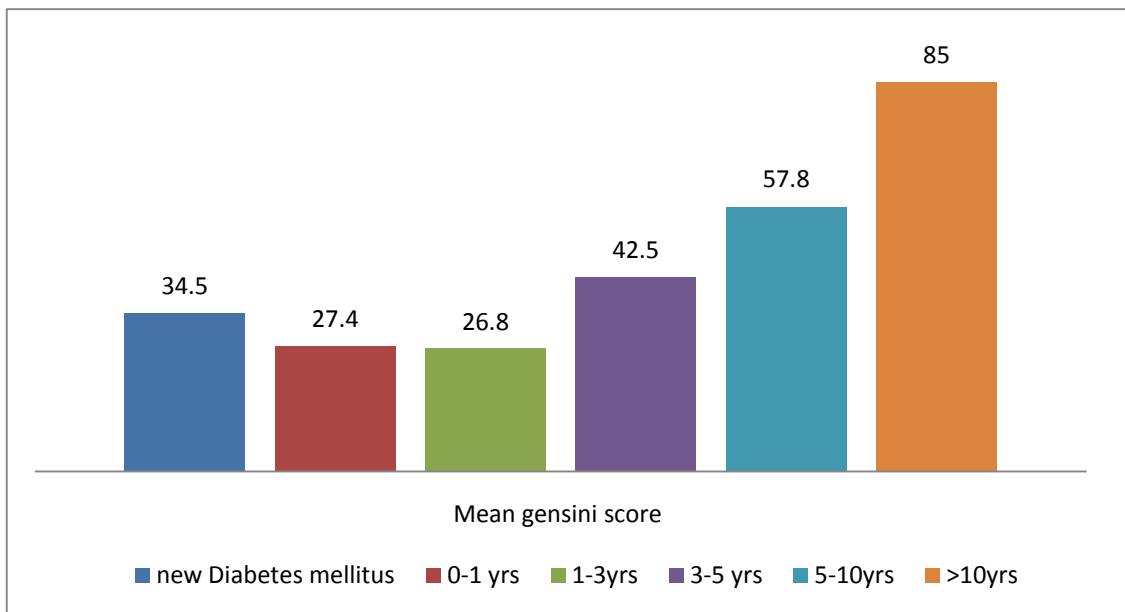
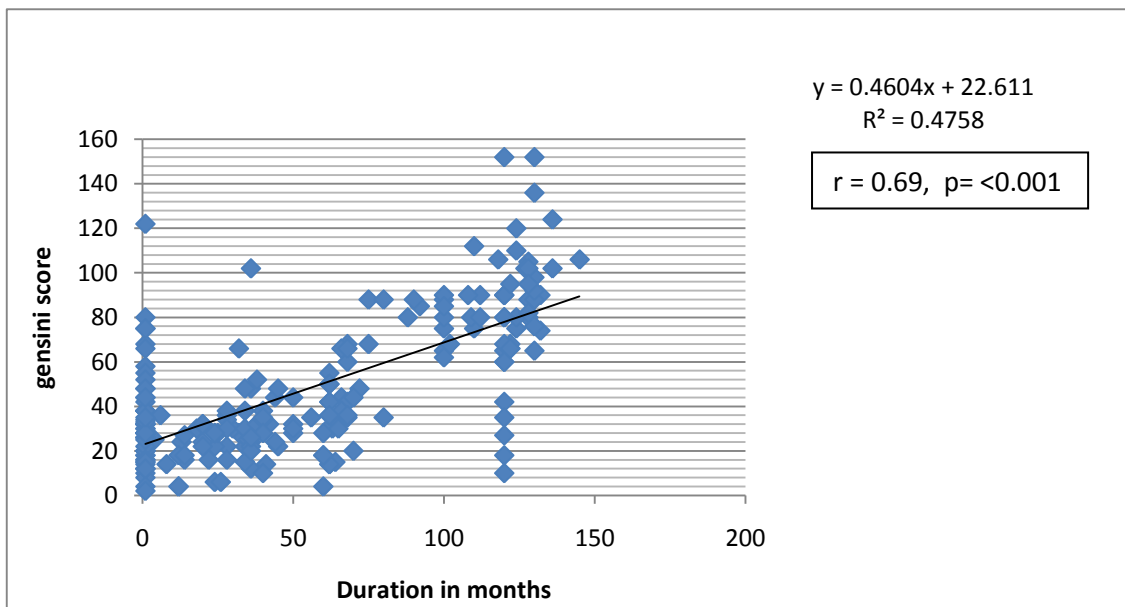


Figure 14: Correlation between duration of diabetes and gensini score in diabetics



ASSOCIATION BETWEEN GLYCEMIC CONTROL (HbA1c) AND GENSINI SCORE IN DIABETICS

Table 13: Association between glycemic control (HbA1c) and gensini score in Diabetics

Glycemic control	hbA1c	Total cases	Mean gensini score	P value
Good	<6.5	14	53.2± 27.8	0.484
Suboptimal	6.5-8.9	141	45.1 ±30.9	
Poor	>9	58	50.4 ±31.6	

There was no statistically significant correlation with the glycemic control as assessed by HbA1c levels to the severity of CAD.

Figure 15: Correlation between HbA1c and gensini score in diabetics

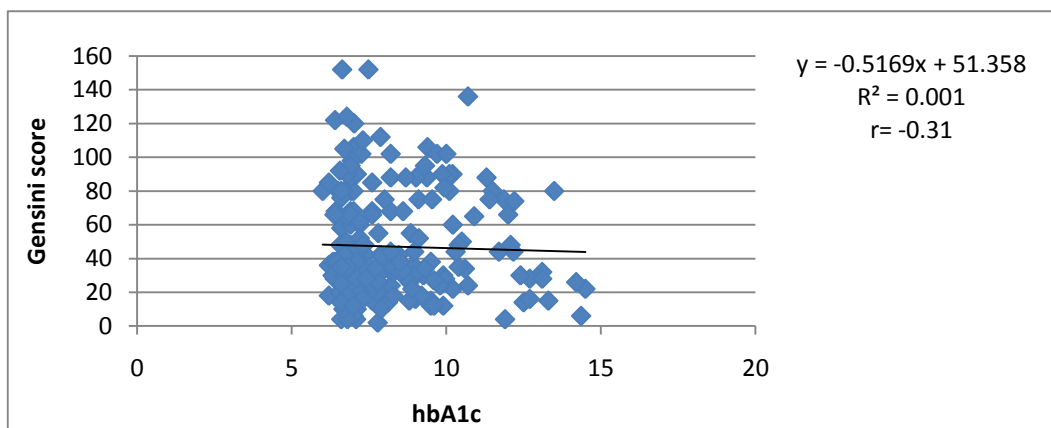
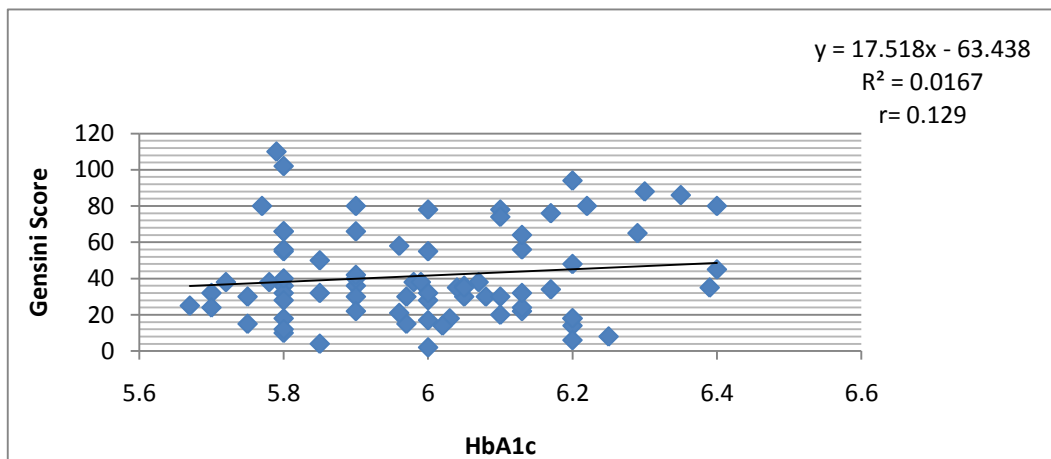


Figure 16: Correlation between HbA1c and gensini score in prediabetics



BLOOD SUGAR LEVELS AMONG THE GROUPS

Table: 14 Blood sugar levels among the groups

Group	Mean Random blood sugar (mg/dl)	Mean Fasting blood sugar (mg/dl)	Mean post prandial blood sugar (mg/dl)
Diabetics	205±86.7	150.9±58.5	188.5±55.3
Prediabetics	134.7±27.4	110.3± 13.38	148.9±16.6
Non diabetics	120± 31.7	111.35±93.69	134.1±17.11

CORRELATION COEFFICIENT BETWEEN SUGAR LEVELS AND GENSINI SCORE

Table 15: Correlation coefficient between sugar levels and gensini score

Group	Random blood sugar (mg/dl)	Fasting blood sugar (mg/dl)	Post prandial blood sugar (mg/dl)
Diabetics	0.0001	0.0079	0.0043
Prediabetics	0.211	0.179	0.127
Non diabetics	0.069	0.012	0.008

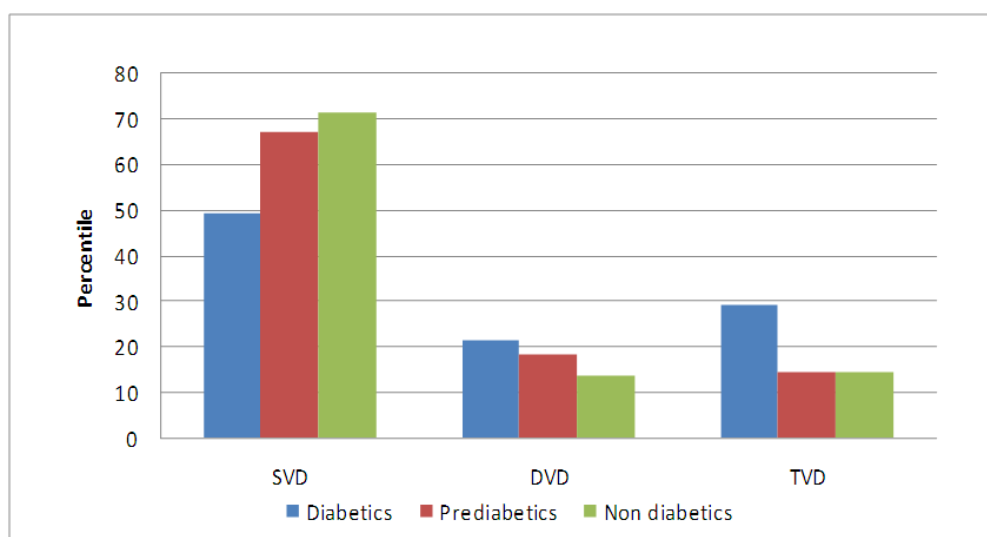
The mean blood sugar levels at admissions were higher in the diabetic group. There was a direct positive correlation between blood sugar levels and the gensini score in all the 3 groups.

CORONARY ARTERY INVOLVEMENT IN DIFFERENT GROUPS

Table 16: Coronary artery involvement in different groups

	Single vessel	%	Double vessel	%	Triple vessel	%	P value
Diabetics	105	49.3	46	21.6	62	29.1	<0.001
Prediabetics	51	67.1	14	18.4	11	14.5	
Non Diabetics	88	71.54	17	13.8	18	14.6	

Figure 17: Coronary artery involvement in different groups



Presence of Triple vessel disease was 29.1% in diabetics and 14.5% and 14.6% among prediabetics and non diabetics respectively. Single vessel disease was seen more in non-diabetics. Single vessel disease was found in 71.54% of non diabetics and 49.3% of diabetics. Triple vessel disease was more common in diabetics, whereas single vessel disease was more common in non-diabetic and pre-diabetics. These observations were statistically significant with a p value of <0.001.

DISCUSSION

A total of 412 patients admitted with first time acute coronary syndrome during the study period were analysed. Of which 213 patients were diabetics and 123 were non-diabetics and 76 were pre-diabetics. Majority of the patients were diabetics. We compared the mean age, sex, risk profile, lipid abnormalities, clinical profile, type of ACS among each group. Correlation between glycemic status according to FBS, PPBS, HbA1c levels and severity of CAD assessed by gensini score were analysed. As with other studies diabetics were the majority in our study.

AGE AND SEX DISTRIBUTION

The mean age of presentation in diabetics was 56.3 ± 10.2 years, non-diabetics 54.5 ± 12.2 years, and pre-diabetics 54.4 ± 9.3 years. There was no much difference in the mean age of presentation in the study groups. In all the 3 groups most of the patients were in the age range between 46 to 60 years. In patients less than 45 years, non-diabetics were more affected than diabetics. In the *GUSTO-1*^{III} trial, it was observed that diabetic patients were older when compared to non diabetic patients, similar results were observed in our study.

Males were more affected in all the three groups, with the highest noted in non-diabetics with a sex ratio of 6.23:1 and lowest ratio in diabetics with 4.46:1. It is obvious that diabetic females had more prevalence of CAD when compared to pre-diabetics and non-diabetics. Pre-menopausal females in general have protection against IHD, but this cardio protection is lost in the presence of diabetes.

SYMPTOM ANALYSIS

Chest pain followed by sweating was the predominant symptom in all the 3 groups. Atypical symptoms such as abdominal pain, giddiness were higher in the diabetic group.

In a study by Soler et al¹¹² diabetic patients had more atypical presentations of ACS with heart failure, vomiting, collapse, confusion or CVA, which led to increased risk for them going undiagnosed and increased mortality.

PATTERN OF ACS IN STUDY POPULATION

In all the 3 groups, ST elevation myocardial infarction was the most common type of ACS. STEMI was particularly higher in the non-diabetic (69.1%) and pre-diabetic group (61.9%). This may indirectly denote that prothrombotic mechanisms may be responsible for ACS among these groups. NSTEMI and unstable angina were higher in diabetic patients. Of the STEMI distribution, anterior wall MI was the most common type followed by inferior wall myocardial infarction in all the three groups.

RISK FACTORS:

Of the risk factors studied, hypertension was more prevalent in the diabetic group. 49.7% of diabetic patients had hypertension. There was no much difference in the prevalence of hypertension among pre-diabetics and non-diabetics. Similar higher prevalence of hypertension among diabetics was noted in other studies (GUSTO-I, *Rancho Bernardo*, *UKPDS*, Gokhroo et al)¹¹¹⁻¹¹⁴

Prevalence of smoking was also high in the diabetic group. Obesity was higher in both pre-diabetic and diabetic patients. Obesity was observed in 47.4% and 46.6% of pre-diabetic and diabetic patients respectively¹¹⁵.

LIPID ABNORMALITIES

In comparing the total cholesterol levels, 19% of diabetics and 18% of pre-diabetics and 18% of non-diabetics were in the borderline high risk category. Higher levels of total cholesterol were observed in 10%, 11%, 6% of patients in diabetic, pre-diabetic and non-diabetic groups respectively.

In comparing the HDL levels, HDL levels were lower in 69% and 73% of diabetics and pre-diabetics respectively. Whereas, only 63% of patients in non-diabetic group had low HDL levels.

Hypertriglyceridemia was noted to be higher in the pre-diabetic group (17%), whereas only 13% of diabetic patients and 11% of non-diabetic patients had high triglycerides (200-499mg/dl). Very high levels i.e. levels more than 500mg/dl was noted in 2 diabetic patients (1%) and 1 non diabetic patient (1%).

In comparing LDL levels, percentage of patients with higher levels (i.e. LDL 160-189mg/dl) in diabetic, pre-diabetic, and non-diabetic groups were 14%, 8% & 13% respectively. Very high levels (i.e. >190mg/dl) were observed in 9% of pre-diabetics, followed by 6% in diabetic group and 3% in non-diabetic group. A role for LDL in diabetic patients in promoting coronary heart disease in recent clinical trials, e.g., the Scandinavian Simvastatin survival Study (4S)^{116,117}, the Cholesterol and recurrent Events (CARE) trial^{118,119}, and the Long-Term Intervention with Pravastatin in Ischemic Disease (LIPID)¹²⁰ has been well established. All these trials have shown that aggressive LDL-lowering therapy reduces recurrent CHD events in patients with diabetes.

SEVERITY AMONG DIABETICS:

Mean HbA1c levels in diabetics was 8.23 ± 1.88 , and 6 ± 0.19 in pre-diabetic groups. We have used gensini score to estimate the severity of coronary artery disease. This gives a greater detail of assessment of CAD and does not ignore trivial lesions. Severity of CAD was higher in diabetics with a mean gensini score of 47.1 ± 31.1 , followed by pre-diabetics with a mean score of 41.5 ± 25.1 . Non diabetic patients had lesser severity of CAD with a mean score of 38.5 ± 33.9 . Our study is consistent with other studies, depicting higher severity of CAD in diabetics than in non diabetics. Results of other studies are shown in table

Table 17: Similar studies comparing severity of CAD

Principle investigator	Number of cases in study	Results
Calton et al ¹²¹	75	Higher score, more three vessel
Natali et al ¹²²	2253	Higher score, greater effect on female patients
Hamby et al ¹²³	100	Higher score, more three vessel
Pajunen et al ¹²⁴	57 IDDM patients	More severe extensive and distal disease
Thomas et al ¹²⁵	59	More severe disease
Abadie et al ¹²⁶	36	No difference
Krishnaswami S et al ¹²⁷	516	More severe disease and more multivessel disease
Mahdi moosavi et al ¹²⁸	50	More severe disease and more multivessel disease
Peppes et al ¹²⁹	115	More severe disease

SEVERITY BY NUMBER OF VESSELS

Multi vessel involvement was more in diabetics. 29.1% of diabetics had triple vessel disease, whereas only around 14% of pre-diabetic and non-diabetic patients had triple vessel disease. 71.5% of non-diabetic patients and 67.1% of pre-diabetic patients had single vessel disease, whereas only 49.3% of diabetics presented with single vessel disease. Pre-diabetics also had a trend towards single vessel disease. There are a number of studies showing higher involvement of multi vessel disease in Diabetes^{123,127,128}. Similar results have been found in other studies using gensini score¹²¹⁻¹²³.

SEVERITY AMONG PREDIABETICS

In our study we have quantitatively assessed the severity of CAD by using gensini score. There are hardly any studies available in pre-diabetics to postulate the extent of severity of CAD. Our study did not show any significant difference in the severity of CAD among pre-diabetics and non-diabetics. It has been well studied and proved that diabetic patients have more severe CAD. But no such major studies are available with pre-diabetic patients to show a higher severity of CAD. Of the pre-diabetic group in our study, 57 cases (75%) had impaired fasting glucose, 58 cases (76.3%) had impaired glucose tolerance and 43 cases (56.5%) had both impaired fasting glucose and impaired glucose tolerance. There was no statistically significant difference in severity of CAD among these groups. Comparatively patients with isolated IFG had a higher gensini score. However patients with IFG, IGT and combined IFG & IGT had higher scores compared to non-diabetics. Yan et al¹³⁰ showed higher severity of CAD in pre-diabetics compared to diabetics, However in his study patients with combined IFG & IGT had more severe disease compared to isolated IFG & IGT.

Pre-diabetic patients are at increased risk of death due to CAD¹³¹. This is chiefly attributed to the fact that most of the ACS in pre-diabetic patients was due to thrombus formation at sites of a vulnerable plaque. The first abnormality noted in patients with insulin resistance is IFG (or impaired glucose tolerance). The presence of IFG usually accompanies long-standing insulin resistance. Many prospective studies^{132,133} show that IFG or IGT is a risk factor for CVD, the degree of independence as a risk factor, however, is uncertain.

DURATION OF DIABETES

Of the 213 diabetic patients, 56 patients were newly diagnosed to have diabetes. The mean duration of diabetes was 53 months in the diabetic group. It was observed that the severity of CAD was higher in patients who had longer duration of diabetes. Patients with long standing diabetes (i.e >10 years) had more severe CAD with a mean score of 85. There was a positive linear correlation between the duration of diabetes and severity of CAD. This was statistically significant. [Correlation coefficient $r = 0.69$, $p = <0.001$]. Studies by Tahir et al¹³⁴ & Syvanne et al³⁵ also found a positive linear correlation with duration of diabetes and severity of CAD.

GLYCEMIC CONTROL

Diabetics with good glycemic control (i.e. HbA1c level <6.5) had a mean gensini score of 53.2 ± 27.8 . Patients with suboptimal control (HbA1c 6.5-8.9) and poor control (HbA1c >9) had a mean gensini score of 45.1 ± 30.9 and 50.4 ± 31.6 respectively. But this was statistically insignificant. When comparing the association between HbA1c levels to the severity of CAD in diabetic patients, there was no statistically significant correlation between them. But among pre-diabetics there was a positive linear correlation identified between the HbA1c levels and gensini score [correlation coefficient $r = 0.129$, $p = <0.01$]. Tahir¹³⁴ and

Ayhan et al¹³⁶ had found positive linear correlation between hbA1c levels and severity of CAD in diabetes patients, our study did not show any relation between them.

The mean blood glucose levels at admission were higher in the diabetic group with 205 ± 86.7 mg/dl. Similarly mean fasting blood sugar levels were also higher in the diabetic group. 21 non diabetic patients had blood sugar levels >140 mg/dl at admission, which could be due to stress hyperglycemia. There was a significant positive linear correlation between FBS, PPBS, presenting RBS and gensini score in all the 3 study groups. This implies that hyperglycemia is directly associated with more severe CAD.

LIMITATIONS OF STUDY

1. Number of patients in the study is small. Hence more studies on pre-diabetics and non-diabetics are needed in future
2. Duration of study is short, Hence follow up of patients was not included in the study
3. Possible underlying mechanism (such as insulin resistance, pro-thrombotic work up) in Pre-diabetic patients was not studied. Large prospective studies in pre-diabetic patients are needed in future for better understanding and planning management strategies.

CONCLUSION

1. A total of 412 patients were studied, of which majority of the patients were diabetics (51.7%)
2. Male predominance was noted in all the study groups
3. Mean Age of presentation was 56.3 ± 10.2 among diabetics, 54.5 ± 12.2 among non-diabetics and 54.4 ± 9.3 among pre-diabetics. There was no much difference in mean age of presentation among study groups. Patients with age range between 46-60 years were commonly affected in all the study groups.
4. Chest pain and sweating were the common presenting symptom in all groups
5. STEMI was comparatively higher in non-diabetics. However this was statistically insignificant.
6. Hypertension was more among diabetics, whereas obesity was higher in both Pre-diabetics and diabetics.
7. Diabetics had more prevalence of Low HDL. 9% and 6 % of Pre-diabetics and diabetics had very high LDL levels respectively.
8. Diabetics had more severe CAD with a gensini score of 47.1 ± 31.1 , followed by Pre-diabetics with a gensini score of 41.5 ± 25.1 . Non-Diabetics had comparatively less severe CAD with a gensini score of 38.45 ± 33.9 .
9. Single vessel disease was more in pre-diabetics and non-diabetics. Whereas diabetics had more triple vessel disease.
10. There was no significant difference in the severity of CAD among Pre-diabetic patients with IFG alone, IGT alone and combined IFG and IGT.
11. Patients with diabetes had a significant positive linear correlation between duration of diabetes and severity of CAD.

12. There was no statistically significant correlation between HbA1c levels and severity of CAD in diabetics.
13. Presenting blood sugar levels was found to have significant positive linear correlation with severity of CAD in all the three groups. This finding was more consistent in pre-diabetic patients.

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ANNEXURE I

LIST OF ABBREVIATIONS USED

CAD	: Coronary Artery Disease
DM	: Diabetes Mellitus
NIDDM	: Non Insulin Dependent Diabetes Mellitus
NCEP	: National Cholesterol Education Program
ATP	: Adult treatment panel
VLDL	: Very Low Density Lipoprotein
LDL	: Low Density Lipoprotein
HDL	: High Density Lipoprotein
HT	: Hypertension
HbA1C	: Glycosylated Haemoglobin
CVD	: Cardiovascular disease
ACS	: Acute coronary Syndrome
ADA	: American diabetes association
GDM	: Gestational diabetes mellitus
FBS	: Fasting blood sugar
PPBS	: Post prandial blood sugar
OGTT	: Oral glucose tolerance test
RBS	: Random blood sugar
MI	: Myocardial infarction
cTn	: Cardiac Troponin

URL	: Upper reference limit
LBBB	: Left bundle branch block
PCI	: Percutaneous coronary intervention
ECG	: Electro cardiogram
CABG	: Coronary artery bypass graft
IFG	: Impaired fasting glucose
IGT	: Impaired glucose tolerance
FFA	: Free fatty acid
VSMC	: Vascular smooth muscle cell
CHD	: Coronary heart disease
NO	: Nitric oxide
STEMI	: ST elevation myocardial infarction
NSTEMI	: Non ST elevation myocardial infarction
IHD	: Ischemic heart disease
CVA	: Cerebro-vascular accident
VCAM	: Vascular cell adhesion molecule
PAI	: Plasminogen activator inhibitor

ANNEXURE II

Case Proforma:

Name : Age : sex:

Op.no: IP.no: unit:

D.O.A: D.O.D:

Duration of stay:

Diagnosis:	AW	IW	LW	STEMI / NSTEMI
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WP: Killip :

HISTORY

Typical/ atypical presentation of CAD: chest pain, dyspnea, sweating, abdomian pain, syncope)

DM : Yes / No Duration : yrs 0-3 , 4-7, 7-10, >10 Newly detected

Treatment details : Not on treatment / Diet / OHA / OHA + insulin / Insulin

Risk factors : smoking (yrs) / family history / tobacco chewing / DLP / HTyrs

History of Diabetic / hypertensive complications :

Other significant history

Clinical Examination

PR: BP: RR:

Ht:	Wt	BMI	WH ratio
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CVS – Features of LV dysfunction : present / absent

ECG : STEMI AW IW LW PW NSTEMI

ECHO : RWMA : LAD / LCX / RCA / Global LV EF : <30 / 30-40 / 40-50 / >50

Investigations :

Hb Ur Cr K+ Trop T

FBS	PPBS	HbA1c:	<5.7	5.7-6.4	6.5-7.5	>7.5
100	100	5.7	100	100	100	100
100	100	6.4	100	100	100	100
100	100	7.5	100	100	100	100
100	100	8.6	100	100	100	100
100	100	9.7	100	100	100	100
100	100	10.8	100	100	100	100
100	100	11.9	100	100	100	100
100	100	13.0	100	100	100	100
100	100	14.1	100	100	100	100
100	100	15.2	100	100	100	100
100	100	16.3	100	100	100	100
100	100	17.4	100	100	100	100
100	100	18.5	100	100	100	100
100	100	19.6	100	100	100	100
100	100	20.7	100	100	100	100
100	100	21.8	100	100	100	100
100	100	22.9	100	100	100	100
100	100	24.0	100	100	100	100
100	100	25.1	100	100	100	100
100	100	26.2	100	100	100	100
100	100	27.3	100	100	100	100
100	100	28.4	100	100	100	100
100	100	29.5	100	100	100	100
100	100	30.6	100	100	100	100
100	100	31.7	100	100	100	100
100	100	32.8	100	100	100	100
100	100	33.9	100	100	100	100
100	100	35.0	100	100	100	100
100	100	36.1	100	100	100	100
100	100	37.2	100	100	100	100
100	100	38.3	100	100	100	100
100	100	39.4	100	100	100	100
100	100	40.5	100	100	100	100
100	100	41.6	100	100	100	100
100	100	42.7	100	100	100	100
100	100	43.8	100	100	100	100
100	100	44.9	100	100	100	100
100	100	46.0	100	100	100	100
100	100	47.1	100	100	100	100
100	100	48.2	100	100	100	100
100	100	49.3	100	100	100	100
100	100	50.4	100	100	100	100
100	100	51.5	100	100	100	100
100	100	52.6	100	100	100	100
100	100	53.7	100	100	100	100
100	100	54.8	100	100	100	100
100	100	55.9	100	100	100	100
100	100	57.0	100	100	100	100
100	100	58.1	100	100	100	100
100	100	59.2	100	100	100	100
100	100	60.3	100	100	100	100
100	100	61.4	100	100	100	100
100	100	62.5	100	100	100	100
100	100	63.6	100	100	100	100
100	100	64.7	100	100	100	100
100	100	65.8	100	100	100	100
100	100	66.9				

Oral GTT : 0 hr = 120 mins :

Chol:

TG:

HDL:

LDL:

Angiography : LMCA / LAD / LCX / RI / RCA /

SVD / 2VD / TVD / LMCA

Gensini score = sum of (stenosis score x functional significance score)	
Significance score	
LM : 5	d-LCx : 1
p-LAD : 2.5	OM : 1
m-LAD : 1.5	p-RCA : 1
d-LAD : 1	m-RCA : 1
1 st Dx : 1	d-RCA : 1
2 nd Dx : 0.5	PD : 1
p-LCx : 2.5	PL : 1
Stenosis score	
1 : 25%	2 : 50%
4 : 75%	8 : 90%
16 : 99%	32 : 100%
LM indicates left main, LAD, left anterior descending; Dx, diagonal; OM, obtuse marginal; RCA, right coronary artery; PD, posterodescending; PL, posteolateral; p-, proximal; m- mid; d- distal	

Gensini score:

0-20	20-40	40-60	60-80	80-100	100-120
120-140	140-160	>160			

|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|

name	ip number	Age	sex	chest pain	dyspnea	sweating	syncope	asymptomatic	giddiness	abd.pain	STEMI	thrombolysed ?	aw	iw	iw+RV	Lateral wall	unstable angina	NSTEMI	Hypertension	duration	Smoking	dm	new Diabetes mellitu	Duration of DM	RBS	FBS	PPBS	2 hr GTT	hba1c	Total cholesterol	TGL	HDL	LDL	EF	vessel	gensini
anthony	i12016411	41	1	1							1	1	1	0	0	0	0	0	1	4		1		88	124	90	155		6.92	152	153	25	143	57	1	80
aravind shanmugam	i12009403	48	1	1		1					1	1	0	0	1	0	0	0	1	2	1	1		36	398	372	399		9.5	251	297	42	163	59	1	12
arjunan	i12025606	41	1	1							0	0	0	0	0	0	0	1	0			1	1	1	126	90	125		7	230	186	59	142	62	2	80
arumugam	i11021280	43	1	1		1			1		1	0	0	1	0	0	0	0	1	2		1		62	235	158	175		8.85	156	142	27	128	53	3	55
arumugam	i11023098	60	1	1		1					0	0	0	0	0	0	1	0	0			1		64	358	193	266		9.5	150	175	23	104	65	1	15
arumugam	i11025132	59	1	1		1					1	1	1	0	0	0	0	0	0			1	1	1	224	109	188		6.4	190	180	34	94	56	3	122
arumugam	i11032892	64	1	1	1	1					0	0	0	0	0	1	0	1	0		1	1		28	113	99	132		6.53	114	115	22	76	81	3	16
ayyavu	i11030361	49	1	1	1						0	0	0	0	0	0	0	1	1	2		1		24	213	144	177		10.21	179	288	30	111	49	1	22
aziz mulik	i11025803	65	1	1							1	1	0	1	0	0	0	0	0			1	1	1	188	90	155		6.42	193	153	27	140	80	2	32
babuji	i12021586	46	1	1							1	0	1	0	0	0	0	0	1	1	1	1		62	224	128	164		8.95	168	222	43	156	59	2	36
balasubramaniam	i11028349	65	1	1		1					0	0	0	0	0	0	1	0	0			1		72	297	135	169		12.08	155	86	37	92	58	3	48
balasubramaniam	i12022051	60	1	1							1	1	0		1	0	0	0	0			1		20	178	99	154		8.2	208	167	61	126	68	1	24
boopathi	i12025313	47	1	1		1					1	1	0	1	0	0	0	0	1	2	1	1		100	201	201	268		9.1	222	476	36	126	60	3	75
chinnappan	i12031013	71	1	1	1	1					1	0	0	1	0	0	0	0	1	3	1	1	1	1	336	265	304		6.9	183	83	54	112	58	1	32
chinnaraj	i11035062	48	1	1					1		0	0	0	0	0	0	0	1	0			1		62	120	120	186		7.4	126	149	27	76	50	1	42
chinnasamy	i11034277	56	1	1		1					1	0	1	0	0	0	0	0	0			1	1	1	225	109	169		6.6	176	85	40	111	69	3	58
chinnasamy	i12015739	71	1	1		1					0	0	0	0	0	0	0	1	0		1	1		13	114	95	145		7	110	125	29	65	56	1	24
deivasigamani	i12016221	62	1	1							1	1	1	0	0	0	0	0	0			1	1	1	143	87	136		6.8	206	144	39	154	79	1	8
deventhiran	i12026953	50	1	1	1	1					1	0	1	0	0	0	0	0	0			1		38	236	156	186		9.26	146	139	25	100	45	1	30
dharmalingam	i12001428	78	1	1							0	0	0	0	0	0	0	1	1	5	1	1	0	62	234	244	275		8.11	107	78	37	57	62	1	14
dhayanan	i12011391	36	1								1	1	1	0	0	0	0	0	0			1		35	72	111	146		7.2	126	145	35	165	66	1	25
dominic	i12018582	48	1	1							0	0	0	0	0	0	0	1	1	5		1		60	258	102	169		6.9	120	85	45	103	55	2	18
duraisamy	i12027178	60	1	1							1	0	1	0	0	0	0	0	1	4		1	1	1	151	133	168		6.2	161	190	28	113	48	1	18
elango	i12013331	45	1	1		1					0	0	0	0	0	0	0	1	1	2	1	1		100	188	188	254		10.1	188	235	113	187	55	3	90
eswaran	i11034646	37	1	1		1					1	p	0	0	1	0	0	0	0			1	1	1	313	111	176		11.4	171	143	32	109	44	1	75
ganesan	i12027570	61	1	1		1					0	0	0	0	0	0	1	0	0			1		62	194	136	255		6.75	190	107	31	146	50	1	50
gopalakrishnan	i12022243	73	1	1	1	1					0	0	0	0	0	0	0	1	1	3		1	0	110	133	114	165		8	218	54	64	157	30	3	75
gopalakrishnan	i12027628	55	1	1							0	0	0	0	0	0	0	1	0			1		18	188	84	154		8.9	146	121	31	93	45	1	30
govindaraj	i12011882	65	1	1		1					1	1	1	0	0	0	0	0	0		1	1		28	91	90	132		6.6	175	145	38	125	45	1	36
gunasekaran	i11034203	39	1	1							1	1	1	0	0	0	0	0	0			1		110	249	135	246		7.87	264	209	37	190	65	3	112
jagajothi	i12000417	66	1	1	1	1					0	0	0	0	0	0	0	1	1	3		1		65	248	156	232		8.96	200	200	33	130	75	3	30
jayaraj	i12002664	52	1	1		1					1	1	1	0	0	0	0	0	0		1	1		60	338	161	224		9.2	230	179	32	169	45	1	18
jayaraj	i12012434	62	1	1		1					0	0	0	0	0	0	0	1	0			1	1	1	106	139	220		7	180	144	36	129	48	3	68
john	i12002729	63	1	1							1	0	0	1			0	0	0		1	1	1	1	189	129	180		7.08	213	86	39	158	58	3	44
kadhirullah	i11023460	62	1	1					1	1	0	0	0	1			0	1	1	10		1		62	332	236	299		10.5	344	367	28	249	57	3	50
kalavendhan	i12013713	46	1	1							0	0	0	0			0	1	1	5		1		120	108	188	222		8.29	281	243	35	204	45	2	18
kaliappan	i12031942	65	1	1	1						0	0	0	0			0	0	0		1	1		136	198	171	190		7.25	120	127	25	78	28	3	102
kalisamy	i12016939	59	1	1							0	0	0	0			0	1	0		1	1	1	1	175	114	166		9.9	189	126	37	136	50	1	30
kamnnaian	i12003556	68	1	1		1					0	0	0	0			1	0	1	2		1		26	158	112	136		7	152	142	25	85	60	1	6
kandasamy	i12001193	49	1		1	1					0	0	1	0			0	0	0			1		24	248	202	266		6.7	138	80	24	125	63	3	28
kannan	i12018589	42	1	1		1					0	0	0	0			0	1	1	2		1	0	92	106	98	155		7.6	141	124	42	105	50	2	85
karuppusamy	i12010492	61	1	1		1					1	1	1	0			0	0	1	2	1	1		25	91	107	123		6.9	163	125	21	125	68	3	28
karuppusamy	i12005782	44	1	1		1					1	1	1	0			0	0	1	5	1	1	1	1	110	103	133		6.6	174	434	25	95	77	1	4
kathirvel	i12029919	60	1	1							1	0	1	0			0	0	0			1		60	168	111	147		6.68	148	84	28	102	50	1	28

name	ip number	Age	sex	chest pain	dyspnea	sweating	syncope	asymptomatic	giddiness	abd.pain	STEMI	thrombolysed ?	aw	iw	iw+RV	Lateral wall	unstable angina	NSTEMI	Hypertension	duration	Smoking	dm	new Diabetes mellitu	Duration of DM	RBS	FBS	PPBS	2 hr GTT	hba1c	Total cholesterol	TGL	HDL	LDL	EF	vessel	gensini
krishnamoorthy	i12015757	61	1	1	1							1	0	1	0		0	0	1	5		1		109	332	290	336		10.1	173	146	42	119	41	1	80
krishnamoorthy	i11025817	62	1	1							0	0	0	0			0	1	0			1		120	104	123	169		6.63	102	101	22	63	60	3	152
krishnan	i12002554	44	1	1		1					0	0	0	0			1	0	0		1	1	1	1	122	101	144		7.11	142	114	33	83	64	1	34
krishnasamy	i12003524	60	1	1							1	0	0	1			0	0	0			1		118	235	216	190		9.39	197	137	25	136	35	3	106
kumar	i12008825	53	1	1		1					1	1	1	0			0	0	0			1		68	299	255	366		7.2	199	204	38	130	46	2	60
kunikam	i12021173	56	1	1		1					0	0	0	0			1	0	1	5		1		34	170	105	155		7.6	165	148	36	148	65	1	15
lakshmanan	i11029760	59	1	1	1	1					0	0	0	0			1	0	1	3		1		122	171	128	165		6.42	199	181	31	124	22	3	68
mahalingam	i11028142	56	1	1					1		1	1	0	1			0	0	0		1	1		66	103	116	136		6.88	123	118	23	74	62	3	38
mahendran	i12014526	55	1	1		1					0	0	0	0			0	1	1	3		1	1	1	260	222	268		7.25	124	195	25	70	65	1	18
mandharchalam	i12023522	83	1	1		1					1	0	0	1			0	0	1	6	1	1		30	199	177	188		6.32	126	53	36	83	62	3	30
mani	i12023907	65	1	1							0	0	0	0			0	1	0		1	1	1	1	136	126	166		8.23	152	262	38	81	56	1	32
manikandan	i12004615	46	1	1							0	0	0	0			1	0	0			1		28	212	193	327		9.3	190	125	35	133	52	2	34
manoharan	i12028027	56	1	1		1			1		1	1	0	1			0	0	1	5		1		34	159	144	165		8	185	142	40	123	50	3	38
manokaran	i11029958	45	1	1		1					1	p	1	0			0	0	0		1	1	1	1	201	188	236		7.2	259	175	32	178	60	1	26
michael raj	i12018578	45	1	1		1					0	0	0	0			0	1	0			1	1	1	209	188	196		6.6	175	186	42	168	56	2	14
mohamad ali	i12007101	66	1	1		1					0	0	0	0			0	1	0			1		68	131	129	114		7.04	133	89	29	93	60	3	36
mohammed	i12024213	59	1	1		1					0	0	0	0			1	0	1	8		1		124	103	111	135		7.3	236	244	48	189	73	3	110
mohammed ali	i12006918	51	1	1							1	1	1	0			0	0	0			1		14	90	86	124		7.6	174	111	28	119	65	2	27
murugaiyan	i12009440	72	1	1	1						1	0	1	0			0	0	1	2		1		122	472	356	224		12	205	139	42	144	30	3	66
murugesan	i12014293	60	1	1		1					1	0	1	0			0	0	1	2	1	1	1	1	321	222	185		9.54	196	142	39	142	57	3	75
murugesan	i12024842	47	1	1		1					1	1	1	0			0	0	1	3		1		28	222	118	148		6.7	140	184	31	86	57	1	30
murugesan	i11030815	53	1	1		1					0	0	0	0			1	0	0			1	1	1	235	201	285		7.8	185	136	57	105	55	2	55
muthusamy	i11035932	76	1	1		1					0	0	0	0			0	1	1	5		1		130	238	255	322		7.48	267	149	30	192	64	3	152
muthusamy	i12003150	76	1	1							1	0	1	0			0	0	0		1	1	1	1	160	106	148		7.2	127	119	20	82	52	2	38
muthusamy	i12011096	64	1	1							1	1	1	0			0	0	1	5		1		34	199	129	185		6.9	169	161	35	112	66	1	30
muthusamy	i12024239	48	1	1		1					0	0	0	0			1	0	1	1	1	1		120	269	221	265		10.9	199	305	45	116	65	3	65
muthusamy gounder	i12012119	75	1			1					0	0	0	0			0	0	0			1		38	134	146	145		7.9	146	139	29	101	56	1	32
nadu gounder	i12012440	75	1	1	1	1					1	0	1	0			0	0	0			1		20	231	336	325		8.3	139	39	44	92	46	1	32
nagaraj	i12000841	44	1	1		1					0	0	0	1			0	0	0			1		12	107	112	148		6.8	114	71	27	74	62	1	4
nagaraj	i120321706	48	1	1							0	0	0	0			0	1	0			1		36	200	185	256		6.7	145	142	35	132	63	1	22
nagoor	i12007052	49	1	1							0	0	0	0			0	1	0		1	1		112	103	122	200		7.09	177	150	34	129	50	3	90
narayanan	i12024807	51	1	1		1					0	0	0	0			1	0	0		1	1		50	352	222	248		11.7	185	175	45	125	69	1	44
nataraj	i11020838	69	1	1		1					0	0	0	0		0	0	1	0			1	1	1	132	225	298		7.78	179	208	40	118	54	1	2
nataraj	i11024796	51	1	1		1					1	1	1	0			0	0	1	2	1	1		120	115	114	158		7.89	187	205	32	122	60	1	10
padmarajan	i11029438	59	1	1		1			1	1	1	1	0	1			0	0	1	8		1		136	149	149	245		6.78	161	130	24	101	60	3	124
padmavathi	i12028216	75	1	1							1	1	0	1			0	0	1	10		1		66	151	144	200		6.45	224	325	36	186	57	2	66
palaanisamy	i12029738	53	1	1							0	0	0	0			0	1	1	5		1		90	206	136	165		6.9	175	142	54	142	68	1	88
palaniappan	i12005868	62	1	1		1					0	0	0	0			0	1	1	10	1	1		120	287	155	165		7.89	88	60	46	32	55	3	42
palanisamy	12004907	74	1	1	1	1					0	0	0	0			0	1	1	15	1	1		130	190	164	154		6.94	134	91	31	84	53	3	98
palanisamy	i12023033	45	1	1							0	0	0	0			0	1	1	8		1		120	229	158	145		7.6	199	114	35	145	56	3	68
palanisamy	i12024862	65	1	1	1						0	0	0	0			0	1	0			1		120	340	83	152		6.9	238	275	40	163	63	3	60
palanisamy	i12030915	60	1	1	1	1			1		1	0	0	1			0	0	1	10		1		130	108	115	102		6.9	127	110	24	80	28	2	65
paramasivam	i11020598	74	1	1		1					0	0	0	0			1	0	1	5	1	1	1	1	212	99	152		7.76	143	274	30	80	70	1	20
perumalsamy	i12027701	67	1	1		1					0	0	0	0			1	0	0			1		132	102	76	99		7.1	180	142	32	130	68	3	90

name	ip number	Age	sex	chest pain	dyspnea	sweating	syncope	asymptomatic	giddiness	abd.pain	STEMI	thrombolysed ?	aw	iw	iw+RV	Lateral wall	unstable angina	NSTEMI	Hypertension	duration	Smoking	dm	new Diabetes mellitu	Duration of DM	RBS	FBS	PPBS	2 hr GTT	hba1c	Total cholesterol	TGL	HDL	LDL	EF	vessel	gensini	
ponnusamy	i11025931	45	1	1		1					1	0	0	1			0	0	0			1	1		50	122	111	148		6.98	163	121	24	116	62	1	32
poovai	i12028198	58	1	1								0	0	0			0	1	1	2			1		120	147	142	175		7.5	196	136	32	147	60	1	35
prabhakaran	i12014048	41	1	1							1	1	1	0			0	0	0			1		70	210	169	185		7.75	182	147	37	133	52	1	20	
prabhakaran	i12021525	52	1	1		1					0	0	0	0			1	0	0			1	1	1	189	105	208		7.35	118	81	28	79	71	3	48	
prabhu	i12009258	43	1	1		1				1	1	0	0	1			0	0	0			1		24	254	156	258		9.96	175	150	30	122	60	1	28	
radhakrishnan	i11022314	61	1	1		1						1	0	0	1		0	0	0	0		1	1	20	245	202	285		7.35	87	76	15	57	62	1	32	
ragavan	i12021356	45	1	1							1	1	0	1		0	0	0	0	0		1	1	1	412	197	206		12.7	227	256	43	155	58	1	16	
raghu kumar	i12013719	53	1	1					1		1	1	0	1		0	0	0	0			1		50	333	272	247		12.7	202	704	37	100	59	2	28	
raj kumar	i12020743	54	1	1							1	1	1	0		0	0	0	1	1		1		50	208	156	185		12.4	188	163	30	140	53	1	30	
rajagopalan	i12015053	71	1	1		1			1	1	1	0	0	1		0	0	0	1	1	1	1		62	96	99	108		6.71	164	98	42	107	53	2	42	
rajamani	i11023197	59	1	1	1	1					1	0	1	0		0	0	0	0			1	1	1	265	228	285		13.3	322	125	32	182	60	2	15	
rajendran	i12011507	55	1	1							1	0	1	0		0	0	0	1	3		1		120	188	129	165		6.7	204	93	47	153	45	1	80	
rajendran	i12027383	54	1	1							0	0	0	0		0	0	1	1	5		1		128	83	88	155		6.7	126	83	27	82	56	2	105	
raju	i12029503	45	1	1							1	1	1	0		0	0	0	0			1		40	199	102	125		6.67	196	335	28	112	55	1	10	
ramaamoorthy	i12020819	71	1	1		1				1	1	1	1	0		0	0	0	0			1	1	1	276	160	202		6.54	186	492	34	73	54	1	20	
ramachandran	i12032092	79	1	1							0	0	0	0		1	0	0	1	5		1		122	182	122	165		7.6	166	177	32	162	46	3	66	
ramakrishnan	i11021005	66	1	1	1	1					1	1	1	0		0	0	0	0	0		1	1	1	336	165	225		7.8	144	139	29	94	48	1	26	
ramakrishnan	i12021291	56	1	1							0	0	1	0		0	0	1	0			1		62	205	137	189		6.59	173	156	45	102	40	2	36	
ramar	i11031192	46	1	1		1					1	1	0	1		0	0	0	0	0		1	1	1	108	99	108		9.9	165	184	32	112	63	1	12	
ramasamy	i12006902	49	1	1							0	0	0	0		0	0	1	0			1		12	201	155	168		7.2	109	98	34	98	60	1	18	
ramasamy	i12015074	53	1	1		1					0	0	0	0		0	1	0	0		1	1		128	215	144	156		9.02	158	68	35	122	60	1	88	
ranganathan	i12004748	57	1	1							1	0	1	0		0	0	0	1	8		1		112	104	99	111		6	191	61	63	119	55	1	80	
rangaraj	i12014944	54	1	1							0	0	0	0		0	0	1	0		1	1		120	117	124	142		6.8	160	88	31	121	69	1	90	
ravi	i12000776	51	1	1	1	1					0	0	1	0		0	0	0	0			1		130	117	139	169		6.6	198	175	28	107	47	3	76	
robert	i11021108	33	1	1		1					1	0	1	0		0	0	0	0		1	1	1	1	181	175	148		6.4	230	55	49	178	66	1	38	
samiyappan	i12029460	47	1	1							1	0	1	0		0	0	0	1	5		1	1	1	169	133	145		8.81	256	154	46	194	69	1	26	
sampathkumar	i11034668	52	1	1		1					1	0	1	0		0	0	0	1	5		1	1	1	237	156	185		8.46	198	146	28	144	70	2	42	
saravanan	i12015358	37	1	1							0	0	0	0		0	0	1	0			1	1	1	90	99	122		7.5	152	178	35	102	79	1	20	
seetharaman	i12027779	58	1	1							0	0	0	0		0	0	1	0		1	1		44	113	114	136		8.2	245	158	29	164	70	2	44	
sekar	i12001166	41	1	1		1					0	0	0	0		0	0	1	0			1	1	1	257	162	200		9.11	251	546	28	144	70	3	52	
selva rajan	i12030681	64	1	1		1					1	0	0	1		0	0	0	0		1	1		100	205	165	185		6.6	205	41	33	122	60	2	80	
selvaraj	i11024264	43	1	1		1					0	0	0	0		0	0	1	1	8		1		40	114	110	145		7.7	167	122	22	124	80	2	34	
selvaraj	i11034171	61	1	1							0	0	0	0		0	1	0	1	5		1		22	77	88	135		6.9	198	181	32	134	66	2	16	
selvaraj	i11033528	66	1	1							0	0	0	0		0	1	0	1	5		1	1	1	135	110	165		7	174	108	30	122	66	1	22	
selvaraj	i11036200	53	1	1							0	0	0	1		0	0	0	0			1		130	119	137	140		8.2	175	152	37	110	69	3	88	
selvaraj	i12006512	47	1	1							1	1	1	0		0	0	0	0			1	1	1	230	148	185		7.1	206	156	40	196	50	3	38	
selvaraj	i12017742	46	1	1	1	1				1	1	0	0	1		0	0	0	1	4	1	1		108	245	216	201		9.87	215	184	30	161	40	1	90	
selvaraj	i12029693	52	1	1		1					1	p	1	0		0	0	0	0			1		127	399	344	320		9.7	179	82	41	117	45	1	102	
sengotayan	i12010856	59	1	1							1	1	1	0		0	0	0	0		1	1		80	108	98	100		6.8	142	108	25	110	55	2	35	
seniappan	i12020485	55	1	1		1					1	1	1	0		0	0	0	0			1	0	120	388	231	268		13.49	184	54	54	119	60	1	80	
shanmugam.m	i12000873	44	1	1		1					0	0	0	1		0	0	0	1	2		1		122	122	102	145		6.9	196	105	27	145	56	3	95	
shanmugasundaram	i12009419	55	1	1							1	1	1	0		0	0	0	1	2	1	1		100	203	146	202		6.2	175	120	35	169	50	2	85	
sivabalan	i12012491	59	1	1					1		0	0	0	1		0	0	1	1	5		1	1	1	160	142	178		6.45	227	67	47	172	49	3	38	
sivakumar	i12030079	55	1	1		1					0	0	0	0		0	1	0	1	2		1		36	174	94	132		7.2	127	160	31	68	63	1	20	

|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|

name	ip number	Age	sex	chest pain	dyspnea	sweating	syncope	asymptomatic	giddiness	abd.pain	STEMI	thrombolysed ?	aw	iw	iw+RV	Lateral wall	unstable angina	NSTEMI	Hypertension	duration	Smoking	dm	new Diabetes mellitus	Duration of DM	RBS	FBS	PPBS	2 hr GTT	hba1c	Total cholesterol	TGL	HDL	LDL	EF	vessel	gensini
mylathal	i12032121	50	0	1		1						0	0	0			1	0	1	4					100	95	129		5.52	142	62	45	100	56	1	22
pappa	i12008392	43	0	1	1						1	0	1	0			0	0	0						145	108	128		5.5	114	42	45	62	30	3	5
pichiammal	i12018281	50	0	1							1	0	1	0			0	0	0						130	98	125		5.6	206	111	49	113	50	1	6
ranganayaki	i12029401	70	0	1							1	0	1	0		0	0	0	0						132	100	136		5.58	214	73	61	105	50	1	20
sargunam	i11033097	64	0	1		1					1	p	0	0	1	0	0	0	1	1					140	107	148		5.1	154	119	23	110	68	1	42
saroja	i12020608	42	0	1	1						1	1	1	0		0	0	0	1	2					177	141	145		4.9	193	150	34	145	42	1	32
savithri	i11028530	38	0	1		1					0	0	0	0		0	1	0	0						94	98	175		5.6	193	109	36	125	68	1	16
sornam	i12002260	61	0	1	1	1					1	1	1	0		0	0	0	0						147	140	142		5.3	219	108	41	141	49	1	18
viyayalakshmi	i12014241	49	0	1		1					1	1	1	0		0	0	0	1	4					89	92	110		5.6	202	352	27	142	55	1	12
yaseen	i11027892	39	0	1							1	1	0	1		0	0	0	0						99	99	142		5.4	237	61	38	176	50	2	88
ayyasamy	i12024332	63	1	1							0	0	0	0	0	0	0	1	1	5					102	126	123		5.2	194	118	36	154	48	3	75
balakrishnan	i12003421	58	1	1							1	0	1	0	0	0	0	0	0						182	124	125		5.45	220	316	32	144	61	1	102
balaraman	i12000194	74	1	1		1					0	0	0	0	0	0	0	1	1	10	1				125	106	136		5.5	181	37	46	118	56	1	48
balu	i12026630	55	1	1	1	1					1	1	1	0	0	0	0	0	1	3					102	91	108			178	101	29	140	30	1	90
cbinna karuppan	i12004549	47	1	1	1						1	1	1	0	0	0	0	0	0						140	144	142		5.2	136	48	33	88	48	1	6
chandrasekaran	i12033200	52	1	1							1	0	0		1	0	0	0	1	4					103	96	125		5.24	183	84	37	130	72	1	12
chinnaraja	i11024110	53	1	1		1			1	1	1	0	0	1	0	0	0	0	0		1				129	92	135		5.4	151	97	23	116	62	3	28
dhandapani	i11034508	45	1	1		1					1	0	1	0	0	0	0	0	0						88	99	125		5.5	158	84	32	106	42	1	16
edward	i12016651	52	1	1		1					1	1	1	0	0	0	0	0	0						98	92	104		5.48	225	78	47	173	55	1	16
elaiyappan	i12024107	65	1	1							1	0	1	0	0	0	0	0	0						108	104	185		5.6	159	144	25	126	51	2	80
ellumalai	i11022874	41	1								1	0	1	0	0	0	0	0	0		1				119	96	165		5.3	176	132	29	120	67	1	22
gopal	i12017714	48	1	1		1					1	1	1	0	0	0	0	0	0		1				132	96	134		5.1	152	125	42	95	52	2	22
gopal	i12030042	60	1	1	1						1	1	0	1	0	0	0	0	0						106	99	124		5.15	165	152	25	142	42	2	45
goplasamy	i12011055	48	1			1					0	0	0	0	0	0	1	0	1	1					131	111	126		5.44	156	101	38	108	76	1	18
gopi	i12028602	54	1	1		1					1	1	1	0	0	0	0	0	0						87	102	165		5.2	190	96	28	141	45	2	58
govindaraj	i11022568	64	1	1							0	0	0	0	0	0	0	0	1	0					191	117	145		5.38	165	145	34	112	65	2	60
govindaraj	i12018002	30	1	1	1	1					1	1	1	0	0	0	0	0	0		1				124	105	135		5.5	140	220	14	81	36	1	12
gurusamy	i12019136	60	1	1							0	0	0	0	0	0	0	1	1	2					105	91	148		5.66	183	122	43	132	75	1	20
jagadeesan	i12003041	66	1	1							0	0	0	0	0	0	0	1	0						100	75	105		5.6	147	58	44	93	53	1	8
jayakumar	i12029770	42	1	1							1	0	0	1	0	0	0	0	0						98	101	136		5.45	153	128	41	87	53	1	4
jayaram	i12008184	61	1	1	1	1					1	0	1	0	0	0	0	0	0		1				136	121	128		5.6	187	59	57	128	46	3	183
joseph	i12014747	61	1	1							0	0	0	0			0	0	0						122	98	140		5.66	175	80	46	122	55	1	32
kanagaraj	i11027303	51	1	1							1	1	0		1		0	0	0						99	89	136		5.4	148	229	29	76	70	1	16
kandasamy	i12022316	70	1	1		1					0	0	0	1			0	1	1	5					98	96	185		5.2	176	168	35	165	50	1	36
kandhasamy	i11024373	76	1	1		1					0	0	0	0			0	1	0		1				88	95	132		5.5	148	57	37	97	40	3	28
kandhasamy	i12024514	41	1	1		1					0	0	0	0			0	1	0						125	95	142		5.5	153	238	32	93	54	1	12
kaniaapan	i12011318	78	1	1							1	0	1	0			0	0	1	8					136	105	146		5	118	49	51	55	42	1	88
karuppusamy	i12023887	42	1	1		1					1	0	1	0			0	0	0						231	108	142		5.38	197	151	40	140	58	1	12
kolaindaisamy	i12014936	81	1	1	1						0	0	0	0			0	1	0		1				90	95	142		5.6	169	98	24	145	57	2	25
krishnasamy	i12001823	70	1	1		1					0	0	0	0			0	1	0		1				113	101	136		5.55	189	195	24	165	50	1	12
krishnavelu	i11030421	63	1	1							1	0	0	1			0	0	1	10					141	102	124		5.6	176	138	34	122	50	1	18
kumarasamy	i11030241	50	1	1							1	1	1	0			0	0	1	1					114	98	108		5.4	312	345	22	145	42	3	46
kumarasamy	i12000263	46	1	1		1					1	0	0	0		1	0	0	0		1				132	95	104		5.6	165	115	29	117	48	1	35
leoraj	i11021565	63	1	1		1					1	0	0	1			0	0	0						89	81	136		5	123	77	30	75	50	1	38

name	ip number	Age	sex	chest pain	dyspnea	sweating	syncope	asymptomatic	giddiness	abd.pain	STEMI	thrombolysed ?	aw	iw	iw+RV	Lateral wall	unstable angina	NSTEMI	Hypertension	duration	Smoking	dm	new Diabetes mellitu	Duration of DM	RBS	FBS	PPBS	2 hr GTT	hba1c	Total cholesterol	TGL	HDL	LDL	EF	vessel	gensini
linganathan	i11024022	56	1	1		1					1	0	0	1	0	1		0	0	0					203	100	142		5.3	169	45	35	122	45	1	66
malleswaran	i12001209	23	1	1	1	1					0	0	1	0			0	0	0						83	90	105		5.4	185	175	29	159	70	1	8
mani	i12029238	55	1	1		1					1	1	1	0			0	0	1	12					120	108	136		5.5	142	109	26	90	64	2	65
mani	i120310164	68	1	1							1	0	1	0			0	0	0						81	71	105		5.4	126	89	33	74	67	1	14
manivannan	i12001286	33	1	1							0	0	0	1			0	0	0		1				90	95	108		5.6	121	214	27	65	72	1	6
manivelsamy	i110242320	61	1	1		1					1	1	1	0			0	0	0						116	92	112		5.47	102	109	30	51	77	1	22
manoharan	i12002041	55	1	1							0	0	0	0			0	1	0						145	92	142		5.6	177	146	33	116	63	1	18
manoharan	i12015281	54	1	1		1					1	1	1	0			0	0	0		1				114	93	135		5.24	149	120	39	98	47	1	60
marappan	i12013776	56	1	1							1	1	1	0			0	0	1	1	1				103	92	128		5.8	165	126	36	140	71	1	28
marimuthu	i12009488	79	1	1	1						1	1	1	0			0	0	0						140	120	136		5.64	168	57	51	115	60	1	18
marimuthu	i12017998	34	1	1							1	1	1	0			0	0	0						99	92	172		5.46	231	175	34	162	60	1	16
marmuthu	i12027952	49	1	1		1					1	0	0	0			0	0	0						95	98	136		5.1	146	105	42	125	50	2	20
muralishankar	i11030377	50	1	1		1					1	0	1	0			0	0	0						104	95	132		5.3	183	62	33	137	55	3	154
murugesan	i12015155	31	1	1							0	0	0	0			0	1	1	1	1				94	108	142		5.5	172	102	45	102	68	1	2.5
muthu	i12013565	54	1	1	1						1	1	1	0			0	0	0						126	98	136		5.68	204	145	42	153	40	1	32
muthukumarasamy	i11027188	49	1	1							0	0	1	0			0	1	1	2	1				92	94	122		5.29	143	101	24	94	66	1	15
nanjappan	i12023689	68	1	1		1					0	0	1	0			0	1	1	5					102	89	125		5.6	168	88	42	142	72	1	28
nanjukutty	i12021388	63	1	1							1	1	1	0			0	0	1	2					108	113	142		5.45	190	179	42	134	64	1	36
nataraj	i11035124	67	1	1							1	0	1	0			0	0	1	1					107	95	136		5.4	95	94	29	44	60	1	32
natarajan	i12001571	48	1	1		1					0	0	0	0			0	1	0						77	86	122		5.4	151	140	21	104	52	2	58
natrajan	i12032699	72	1	1	1						1	1	0	1			0	0	0						99	88	99		5.5	162	63	37	115	46	1	32
palaniappan	i11032481	71	1	1		1					0	0	0	0			1	0	0						89	110	104		5.34	154	51	40	102	88	3	188
palanimuthu	i12022595	48	1	1							1	1	1	0			0	0	0		1				105	94	142		5.2	176	86	66	101	75	1	32
palanisamy	i12013081	53	1	1		1			1		1	1	0	1			0	0	1	1					102	95	127		5.3	164	98	30	150	40	1	20
palanisamy	i12027045	50	1	1					1		1	1	0	1			0	0	0						89	90	136		5.6	176	145	28	16	60	1	88
palanisamy.o	i12004866	46	1	1							0	0	0	0			0	1	0						97	93	98		5.2	170	126	28	119	52	1	74
periyasamy	i12008232	73	1	1	1	1					1	0	0	1			0	0	1	2					154	101	135		5.5	175	158	26	175	33	3	165
periyasamy	i12025563	48	1	1							0	0	0	0			0	1	0						120	97	132		4.5	97	88	48	47	56	1	10
ponnusamy	i12011198	37	1	1							1	1	0	1			0	0	0						126	123	152		5.4	125	70	37	78	58	2	55
ponnusamy	i12029218	55	1	1		1					1	0	1	0			0	0	0						120	95	136		5.5	119	67	26	80	50	1	39
prakash	i12005999	61	1	1							1	1	1	0			0	0	1	2					120	86	111		5.5	156	102	29	132	55	1	4
prakash	i12017001	39	1	1							0	0	0	0			0	1	0		1				120	102	136		5.4	310	333	45	225	58	3	34
pramod	i12027776	41	1	1		1					0	0	0	0			0	1	0						106	98	136		5.1	169	193	33	103	60	1	8
radhakrishnan	i11020932	54	1	1							1	0	1	0		0	0	0	0						90	95	102		5.6	176	96	35	129	67	1	22
rajan	i12021948	61	1	1							0	0	0	0			0	0	1	0					90	120	125		5.5	175	81	38	121	50	3	46
ramachandran	i12018640	62	1	1		1					0	0	0	0		0	1	0	0						104	91	142		5.5	170	81	40	115	62	1	48
ramanathan	i12004527	50	1								0	0	0	0			0	0	0	0					123	161	136		5.6	168	95	41	107	55	1	42
ranganathan	i11024511	79	1	1							1	1	1	0			0	0	0		1				200	102	150		5.4	130	50	22	95	55	1	75
rangasamy	i12004887	74	1	1		1					0	0	0	0			0	0	1	1	2				106	122	136		5.3	138	119	42	80	38	3	75
rangasamy	i12013100	49	1	1	1	1					1	1	1	0			0	0	0	1	3				107	91	142		5.5	226	94	40	166	36	1	80
sakthivel	i12000053	53	1	1							0	0	0	0			0	0	1	1	2				136	106	135		5.46	144	227	25	81	62	1	52
sakthivel	i12012191	45	1	1					1	1	1	1	0	1			0	0	0	0					105	95	136		5.6	185	152	39	185	52	1	28
sam chelladurai	i12002885	35	1	1	1	1					1	1	1	0			0	0	0	0		1			120	90	125		4.9	170	200	26	152	45	1	12
sastha	i11032818	32	1	1							1	0	1	0			0	0	0	1	4				140	121	165		4.6	232	238	45	156	44	1	52

|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|--|

name	ip number	Age	sex	chest pain	dyspnea	sweating	syncope	asymptomatic	giddiness	abd.pain	STEMI	thrombolysed ?	aw	iw	iw+RV	Lateral wall	unstable angina	NSTEMI	Hypertension	duration	Smoking	dm	new Diabetes mellitus	Duration of DM	RBS	FBS	PPBS	2 hr GTT	hba1c	Total cholesterol	TGL	HDL	LDL	EF	vessel	gensini
arumugam	i12011589	52	1	1		1						0	0	0	0	0	0	1	0						145	120	167	164	5.8	136	47	32	105	68	1	18
balthasar	i12007741	58	1	1	1	1						0	0	0	0	0	0	1	0						165	124	152	195	5.9	218	239	41	114	45	2	80
chinnasamy	i12029662	58	1	1								1	0	0	1	0	0	0	0						145	117	137	175	5.79	189	135	32	139	45	2	110
damodaran	i12012358	65	1	1								0	0	0	0	0	0	1	1	2	1				122	120	139	138	5.77	202	117	39	148	66	1	80
dayanand	i12018528	39	1	1		1						1	0	0	1	0	0	0	0						105	95	128	148	5.72	184	150	42	124	75	1	38
dhandapani	i12006932	55	1	1	1							1	0	0	0	0	1	0	0	0					124	109	139	144	5.85	224	182	47	152	42	2	4
duraisamy	i12012754	71	1	1								0	0	0	0	0	0	1	1	5					154	121	158	150	5.96	125	85	29	85	59	3	58
farook	i12012969	45	1	1		1						0	0	0	0	0	0	1	1	8					136	105	136	138	5.75	156	126	23	110	80	1	30
govindasamy	i12028408	61	1	1								0	0	0	0	0	0	1	0		1				166	115	128	179	5.8	145	78	35	128	80	3	66
govindasamy	i120095026	56	1	1		1						0	0	0	0	0	0	1	0						128	116	136	150	6.2	129	68	33	91	65	1	18
jayapaul	i11028570	56	1	1								1	1	0	0	1	0	0	0						165	129	142	139	6.29	166	105	24	120	66	3	65
kanagaraj	i12030623	50	1	1		1						1	0	0	1			0	0	1	5	1			85	107	136	120	5.85	195	234	28	124	60	1	50
kandasamy	i11027690	68	1	1								1	1	1	0			0	0	1	4	1			122	94	108	147	5.98	150	124	27	99	70	3	38
kittusamy	i12009205	57	1	1		1						0	0	0	0			0	1	0					111	122	142	159	6.05	140	218	30	84	65	1	30
krishnan	i12029082	60	1	1	1							1	1	1	0			0	0	0					115	110	138	126	5.8	222	254	35	155	49	2	32
krishnasamy	i12032597	73	1	1								1	1	1	0			0	0	1	4				120	100	140	138	6.22	113	168	27	66	46	1	80
kumarasamy	i12003671	68	1	1								1	1	0	1			0	0	0					124	96	118	141	6.25	145	108	28	106	50	1	8
kuppan	i12030863	45	1	1	1	1						1	0	0	1			0	0	1	3				102	92	140	152	5.9	140	112	36	84	43	2	36
kuppusamy	i12006437	61	1	1					1			0	0	0	0			1	0	1	2				130	116	148	168	5.7	189	99	57	121	62	1	32
leela krishnan	i12020561	57	1	1								1	1	1	0			0	0	1	5				99	76	165	140	6.08	172	92	32	129	59	1	30
loganathan	i12002296	39	1									0	0	0	0			1	0	0		1			125	99	113	136	6	337	273	19	265	70	1	28
malar mani nathan	i12004802	43	1	1					1			1	1	0	1			0	0	0					166	125	166	174	5.96	185	185	28	132	58	1	21
mohamed ali	i11024680	71	1	1		1						1	1	0	1			0	0	0					94	88	118	150	6.13	111	81	21	74	32	3	64
murugesan	i11029109	65	1	1								0	0	0	0			0	1	0					157	118	169	199	6.1	145	120	34	106	71	1	78
muthusamy	i11023259	54	1	1								1	1	1	0			0	0	0		1			119	115	157	180	6.1	159	60	33	113	72	1	74
muthusamy	i12025161	64	1	1		1						1	0	0	1			0	0	1	5				189	113	149	164	5.8	219	51	49	182	30	3	55
nagaraj	i11024318	43	1	1								1	p	0	1			0	0	1	4				188	122	154	166	5.8	209	76	50	136	52	1	102
natarajan	i12006379	61	1	1	1	1						1	1	1	0			0	0	1	5	1			131	125	148	176	6.02	159	122	31	111	44	1	14
natesan	i12027669	50	1	1		1				1		0	0	0	0			0	1	0		1			132	119	153	150	5.9	225	189	38	169	72	1	40
palanisamy	i11028528	46	1	1								1	1	1	0			0	0	1	4				175	120	142	174	5.8	313	180	31	211	60	1	10
palanisamy	i11035223	73	1	1								1	1	0	1			0	0	0					109	124	163	164	6.2	126	59	36	79	48	1	94
palanisamy	i12002852	46	1	1								1	0	0	1			0	0	0					96	110	168	138	5.8	167	219	26	113	62	1	66
pradeep	i12023550	47	1	1		1						1	1	1	0			0	0	0					148	105	147	162	5.8	215	115	35	158	65	3	40
rajendiran	i12002581	39	1	1								0	0	0	0		0	0	1	0		1			104	107	149	182	6.1	148	220	37	102	40	2	30
raju	i12001827	66	1	1	1	1			1			1	0	1	0		0	0	0	1	5	1			214	119	169	194	6.4	161	62	41	101	30	1	80
ram mohan	i12000528	56	1	1		1						1	0	0	1		0	0	0	0					197	114	147	148	6	205	181	28	156	55	3	78
ramachandran	i12004672	43	1	1								0	0	0	0		0	0	1	1	10				97	92	145	148	5.9	212	123	40	175	63	2	42
ramalingam	i12007939	49	1	1	1	1						1	1	1	0		0	0	0	1	1				164	120	158	164	5.9	114	107	31	70	56	1	30
ramasamy	i11028712	56	1	1								0	0	0	0		0	1	0	1	5				125	96	138	142	6	209	99	47	132	70	2	2
ramasamy	i11029951	46	1	1		1						1	1	1	0		0	0	0	0					139	108	138	142	5.99	166	452	22	66	55	1	38
ramasamy	i12003581	58	1	1	1	1						1	0	1	0		0	0	0	0					136	123	158	168	5.75	200	123	28	141	37	1	15
ranganathan	i12006188	58	1	1		1						0	0	0	0		0	0	1	0		1			109	81	144	140	5.7	181	163	24	134	65	1	24
ranganathan	i12016007	45	1	1								1	0	0	0	1	0	0	0	1	4				174	125	185	169	6.4	265	210	40	193	45	2	45
rangasamy	i12007048	47	1	1								1	1	0	1		0	0	0	0	0				106	94	112	142	6.2	110	130	30	59	68	1	14

name	ip number	Age	sex	chest pain	dyspnea	sweating	syncope	asymptomatic	giddiness	abd.pain	STEMI	thrombolysed ?	aw	iw	iw+RV	Lateral wall	unstable angina	NSTEMI	Hypertension	duration	Smoking	dm	new Diabetes mellitu	Duration of DM	RBS	FBS	PPBS	2 hr GTT	hba1c	Total cholesterol	TGL	HDL	LDL	EF	vessel	gensini
rathinagiri	i12016679	54	1	1							0	0	0			0	0	1	1	1					128	100	142	128	6.2	186	140	36	160	58	1	18
ravikumar	i12007009	47	1	1		1					0	0	0	0		0	0	1	1	2	1				126	118	158	148	6	118	143	20	74	54	1	55
sathyamoorthi	i12003450	39	1	1							1	1	1	0		0	0	0	0						125	98	172	144	5.67	155	169	34	90	65	1	25
seeniappan	i12000383	65	1	1	1	1					0	0	0	0		0	1	0	1	5					128	99	166	165	6.35	175	85	28	149	70	3	86
selvaraj	i11035800	51	1	1							1	p	0	1		0	0	0	0						136	96	148	136	6.3	183	84	35	135	71	1	88
shanmugasundaram	i12003058	45	1	1		1					0	0	0	0		0	0	1	0						124	98	136	128	5.9	162	143	32	102	52	1	22
shanmugavel	i11028471	61	1	1		1					0	0	0			0	0	1	0						136	120	162	175	6.04	161	72	41	94	74	2	35
shanmugavel	i11030574	36	1	1							1	1	1	0		0	0	0	0						151	103	188	169	5.85	150	163	23	98	64	1	32
sivagnanma	i12012472	43	1								0	0	0	0		0	0	1	0		1				128	114	147	155	6	125	48	35	81	66		32
subramani	i12009459	64	1	1							1	1	1	0		0	0	0	0		1				112	109	155	168	6.05	111	103	36	59	40	1	36
subramaniam	i12018447	43	1	1	1	1					1	0	0	1		0	0	0	0		1				122	100	150	147	5.8	100	237	47	147	72	2	56
swamippan	i12024464	63	1	1							1	1	1	0		0	0	0	0						129	115	165	137	6.05	145	110	38	96	42	2	35
thirunavukarsu	i12009097	47	1	1		1					1	1	0	1		0	0	0	1	1					147	119	175	148	5.78	197	215	35	136	40	3	38
ubidur rahman	i12017332	55	1	1							1	0	1	0		0	0	0	0						136	91	136	140	5.8	115	201	26	65	72	1	28
velusamy	i12009465	66	1	1		1					1	0	0	1		0	0	0	0						128	100	155	162	6.2	111	102	25	71	55	1	6
vincent	i12006575	60	1	1		1					1	0	1	0		0	0	0	1	2					118	119	147	138	6.13	269	163	50	198	25	1	32



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ANGIOGRAPHIC SEVERITY OF CAD IN PATIENTS WITH ACUTE CORONARY

BY GIRISH DEEPAK MANOGARAN 16101851 D.M. CARDIOLOGY

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ANGIOGRAPHIC SEVERITY OF CAD IN PATIENTS WITH ACUTE
CORONARY SYNDROME IN CORRELATION TO THEIR GLYCEMIC STATUS

INTRODUCTION

Atherosclerotic vascular diseases, which comprises coronary heart disease and cerebro-vascular disease is a major global health burden. They constitute 21.9 per cent of total deaths globally and are projected to increase further to 26.3 per cent by 2030¹.

15

The prevalence of diabetes is a global health burden. The overall prevalence is expected to rise from 285 million in 2010 to 438 million by the year 2030². While diabetes poses a huge economic burden to all nations, developing countries bear the highest burden since more than 80% of cases occur in these countries.

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